(This number is composed of two sections, of which this is Section I)

Minnesota State Medical Association
Annual Meeting, Minneapolis, May 23-25, 1955

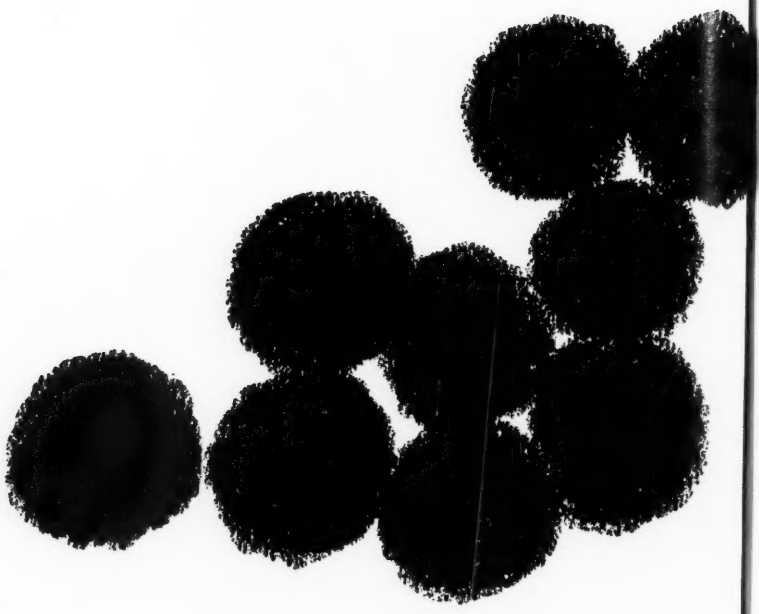
Minnesota MEDICINE

PUBLISHED MONTHLY BY THE MINNESOTA STATE MEDICAL ASSOCIATION

Volume 38 MARCH, 1955 Number 3

Minneapolis Academy of Medicine Number

Printed in U.S.A. (Table of Contents—Page iii) 40c a copy—$3.00 a year



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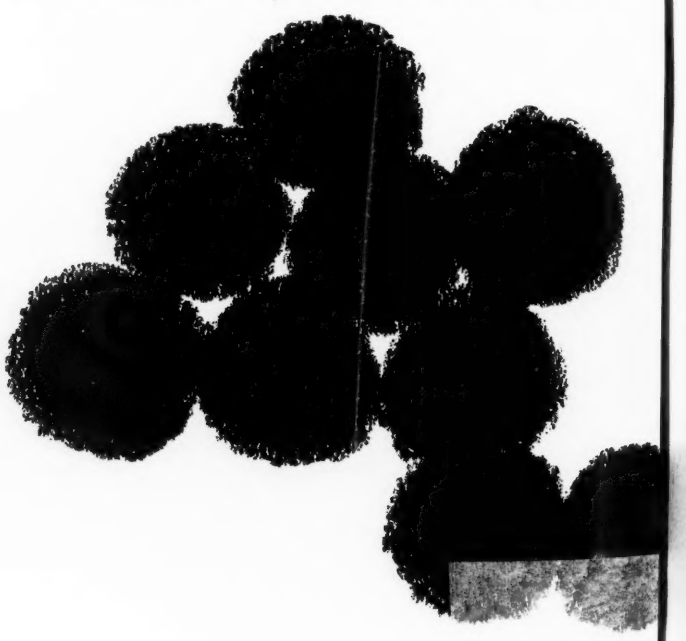

OFFICIAL JOURNAL
Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine, Minneapolis Surgical Society, Minnesota Obstetric and Gynecologic Society, Duluth Surgical Society, and Minneapolis Academy of Medicine.

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OFFICIAL JOURNAL OF THE MINNESOTA STATE MEDICAL ASSOCIATION
496 Lowry Medical Arts Bldg., Saint Paul 2, Minnesota

BUSINESS MANAGER
R. R. ROSELL

Annual Subscription—$3.00. Single Copies—$0.40. Foreign and Canadian Subscriptions—$3.50.

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Original Contributions

Medical Aspects of Peptic Ulcer

JAMES MYHRE, M.D.
Minneapolis, Minnesota

PEPTIC ULCER is a sharply circumscribed loss of tissue resulting from the digestive action of acid gastric juice. It is clearly differentiated from an erosion in that peptic ulcer is a penetrating process involving the muscularis mucosa as well as mucosa.

It has been estimated from autopsy statistics that ten per cent of all individuals at some time or another in their lives have suffered from a chronic gastric or duodenal ulcer. The duodenal ulcer occurs about four to one in the male whereas gastric ulcer occurs three or four to one in the female. A further breakdown as to sex includes approximately only a two percent incidence of perforation of peptic ulcers in the female.

Peptic ulcer may occur in the lower esophagus, stomach, upper portion of the duodenum, in the small bowel when it is anastomosed to an acid-bearing section of the gastrointestinal tract, and very rarely in a Meckel's diverticulum in the jejunum or ileum. In the latter instance it occurs with ectopic gastric mucosa. The vast majority are in the first one to one and one-half inches of the duodenum or the duodenal bulb.

Etiology is a big subject but certain elementary things seem very definite and also pertinent as far as understanding the problem of treatment is concerned. It appears to be simply a digestion of tissue by the acid gastric juices. The old dictum of "No acid, no ulcer" seems incontestable. Peptic ulcer occurs only in those portions of the gastrointestinal tract exposed to the action of the acid gastric juice, and peptic ulcer does not occur in the complete absence of acid gastric juice. The psyche has been increasingly incriminated. There is at present a strong association of peptic ulcer with tension, nervous instability, fatigue and worry. Frequently the emotional aspects in etiology are lacking in gastric ulcers. The psychoanalyst finds strong dependent wishes which may be overt or not at all obvious in these people. In the group with hidden dependency these wishes are rejected by the patient's conscious personality and frequently overcompensated for in a capable energetic attainment of social or economic success. It has been said that a person could have a rousing business if he himself had a good ulcer and had twelve ulcer patients working for him. Harry Truman may have been nearer the core of things than he realized when he spoke of a three-ulcer man in a four-ulcer job. It has been said that hyperthyroidism originated from stress in the home or family and an ulcer originates from stress with external environment, e.g., a business or job. Wolf and Wolf in their patient, Tom, with a gastric fistula, observed hypersecretion during tense anxiety and noted then the transition from a mucosal erosion into an ulcer by the digestive action of acid gastric juice. During the Blitz in London there was a very definite increase in perforation of ulcers. Patients with chronic recurring ulcer distress frequently correlate an exacerbation with fatigue, stress, or tension.

The patient generally gives a history which is rather definite. He might describe the pain as burning, hurting, gnawing, annoying, or aching or just a dull distress or an awareness of discomfort. Sometimes the word "hunger pain" is used but actually hunger pain is an intermittent type of distress whereas ulcer distress is steady and continuous from fifteen minutes up to one or more hours. The presence of a pain in the back suggests chronic perforation. Chronicity of pain is generally characteristic for most patients with an

Presented before the Minneapolis Academy of Medicine, April 19, 1954.

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ulcer for they experience their pain over a period of years. Histories of twenty to thirty years of distress are not uncommon but the common incidence is stated as being between five and eight years. Of course, one can find asymptomatic peptic ulcer or peptic ulcers of very brief duration as only a few days or few weeks. Generally one can bring out or the patient will spontaneously volunteer a daily rhythm to his distress. If the patient is given time and encouraged to give a "typical day," he will frequently describe the distress as coming on one to four hours after eating and he will get relief by eating, drinking milk, or taking soda. Vomiting and rest frequently help. Frequently he will describe waking with distress between midnight and two o'clock in the morning. This suggests pyloric obstruction and/or an acutely inflamed ulcer with a very high gastric secretion. The periodicity of ulcer distress is quite interesting and is classically stated as being in the spring and fall of the year. Many people do notice this periodicity but statistical studies do not bear this out except perhaps in farmers, for the greater distribution is more through the winter months. Periodicity may be correlated with infections, emotional stress, fatigue, or strain.

The final court of appeal is the x-ray or gastroscopic examination. Frequently there is a negative x-ray examination in a patient with a classical ulcer history. In this instance repeating the x-ray will frequently bring out the true state of affairs although there are errors depending on the efficiency of the x-ray examination, body type of the individual, and the difficulty in demonstrating posterior wall lesions. These people should be considered for gastroscopic examination.

When confronted with patients with gastrointestinal distress, part of the job is to decide which patients should have the benefit of x-ray examination. Obviously, the severity of the complaint is considered and whether or not it fits in with what is called classical ulcer distress. In the uncomplicated ulcer, it is quite unusual to have distress before breakfast, and noting this you may be much less inclined to feel an x-ray examination is necessary. The pain of an irritable bowel is much more likely to be intermittent and spread out over the abdomen, and classically the patient will obtain good relief with a good belch or particularly by flatus or a bowel movement. If the patient has hypertension, it is less likely that he has a peptic ulcer. Food relief is strongly suggestive, but soda relief is not entirely conclusive for it produces a belch which can cause relief of functional gaseous distress. Just nausea is uncommon with peptic ulceration, although it is very occasionally the only symptom in a gastric ulcer. A minor pearl is that epigastric distress which disappears with a dark stool is likely due to peptic ulcer. Peptic ulcer is rare in hypoparathyroidism and not uncommon with hyperparathyroidism. It is apparently rare with adrenocortical hyperactivity of the adrenogenital type. Ulcers frequently heal in the first part of pregnancy, and the general incidence in females goes up after menopause. On physical examination, the main finding is rather localized epigastric tenderness over the lesion. On laboratory examination, the gastric analysis is most helpful. "No acid, no ulcer" is most helpful in cataloguing the patient's distress. It is not infrequent, unfortunately, that a gastric analysis shows no free hydrochloric acid and on being repeated two or sometimes three times occasionally will show a plentiful supply of acid. Apparently the tip of the aspirating tube is in the esophagus or is passed into the duodenum. Blood in the stool by Guaiac test is, of course, suggestive of an ulcer with bleeding.

Relief of distress is generally easy. The patient hardly needs a doctor for this but just some advice from an ulcer friend or Aunt Nellie who has taken soda bicarbonate most of her life. Curing an actual ulcer crater is a bigger job, but the real challenge and the real job of the physician is healing the patient's tendency to recurrent ulcer formation or in other words healing a chronic disease. This generally involves changing his reactions to his environment.

Rest is perhaps the most important thing. This is best obtained in a hospital for from two to three weeks, and this is particularly true when the peptic ulcer is first discovered for it is here that the patient will get the best orientation which is so desirable at the onset in his probable chronic disease. It is in this new ulcer group that the best chance for a permanent healing of the disease is found. I have a friend who has a large number of patients apparently of a two to three packages of cigarettes, eight to ten highballs, and sixteen to eighteen hours a day type. They characteristically always have another $25,000 to $50,000 deal coming up the next day, and therefore continually put

off concentrated treatment until the physician, who has considerable stature, has been known to go into their offices and take them by the arm to the hospital. There he pulls the shade, closes the door, orders that there be no telephone calls, no telegrams, no mail, and no visitors (probably particularly the patient's wife). Then with a minimal amount of medication and diet in a matter of a few days, the patient quickly becomes comfortable. Alleviating the chronic tension and fatigue of these people is of the utmost importance and is very nicely abetted by the cheap and practical phenobarbital in sufficient doses so that they can easily succumb to your good advice to never resist an urge to doze. Time should be taken to explain an ulcer to the patient, and this is most easily done with the patient in the hospital. You then can also further observe his reaction to himself, his business, his family, and his environment. I find it most helpful to sit down with a piece of paper and draw out the rough diagram of the stomach, duodenum, small bowel, and colon. Many patients have no appreciation of what the word ulcer means, and some understanding comes for the first time when you compare it to the collar sore on a horse, varicose ulcer on a lady's leg, or a running sore on a dog. Then you can very profitably carry it further by telling them that the acid in their stomach is just like acid in a cream station or acid from their car battery. A previously rather uninterested person becomes rather interested with this explanation and responds rather dramatically to the question, "What do you suppose would happen if battery acid or cream station acid were dropped in the collar sore of a horse for twenty-four hours a day?" He will then understand the value of rest when he realizes that tension and anxiety greatly increase the formation of acid. He will then understand that the frequent feedings are intended to neutralize the acid. He should, of course, entirely and forever quit smoking and drinking. This is not easy to do, of course, but it is best done in the hospital with the help of some sedation. I oversimplify it, but get the point across by telling them about the gastric analysis. I explain that if their gastric analysis showed thirty degrees and the tube were left in place and he were to smoke a cigarette, then the acid if it were aspirated again might show sixty degrees of acid. This makes sense to the patient and makes it much easier for him to quit smoking. Of course, the only way is to quit completely and not to "taper off." Spending this amount of time with the patient convinces him that you are interested, and your interest and enthusiasm are an exceedingly potent weapon in the therapeutic program.

The classical Sippy type of diet has its dating back to antiquity but was organized by Sippy and has very well stood the test of time. Ulcer treatments and ulcer drugs come and go many times a year, and the three to four ounces or half glass of milk and cream stands the test of time. This should be given with regularity on the hour from seven in the morning until seven at night. The half glass is important as it allows physiologic stomach rest, and this, too, should be explained to the patient inasmuch as large feedings cause distention and increased secretions. It is unnecessary in most instances to add antacid. It is expensive and just the milk alone is sufficient with the Sippy diet program of gradually increasing foods like poached or boiled egg, cooked cereal, toast, pureed fruits and vegetables, rice, jello, custard, chicken, and ground beef. In order to impress the patient of the necessity for this diet, you must know about it yourself and be convinced about it. The "three bland feedings a day" has no place in the treatment of peptic ulcer. The drug companies have flooded the market with multiple antacids and minor insignificant variations of them all. When you explain to the patient, as you should, that his ulcer treatment may take anywhere from six months to two years and then figure out what his cost would be for antacids, you will realize that it is not economically desirable. If you elect to add one of the aluminum hydroxide preparations as an antacid, it is probably best done on the half hour from 7:30 in the morning until 7.30 at night. A multiple vitamin preparation should be included on a Sippy diet.

The antisecretory drugs are the great hope in present peptic ulcer investigation and the emphasis in research has swung away from antacids to this type of medication. Banthine is the original and best known of all the drugs, and at the present is preferred because it is so well developed. It is hoped that some of the many brother and sister preparations will have less of the side effects of dryness of the mouth, blurring of vision, and slowing of micturition and above all not to be so variable and unpredictable in their effect. Our

standard procedure is to give one hundred milligrams of Banthine at eight p.m., two a.m., eight a.m., and two p.m, or every six hours around the clock. We have the patient set his alarm clock at two a.m. to do this. At the present time, the antisecretory drugs must just be considered an adjunct to the rest and dietary program. It is rather disconcerting to see some of the sales propaganda encouraging the use of antisecretory drugs alone with three bland meals.

During the daily hospital visits, the various features of ulcer management are discussed with the patient, and he is encouraged to ask questions. The complications of hemorrhage in the form of tarry stools or coffee-ground vomitus should be mentioned. The symptoms of perforation should be impressed on the patient, and the necessity of getting to a physician early should be stressed. Pyloric obstruction should be mentioned. After the orientation in the hospital, the patient should have well in his mind that relief of his distress is easy and, of course, no indication whatever of degree of healing. He should also understand that complete healing is a slow process, and in our patients we do not even bother in the case of a duodenal ulcer to repeat the x-ray for evidence of healing any sooner than three months. The duodenal ulcers heal slowly, and the patients also are a little inclined to relax if there is no x-ray evidence of a crater. It is impossible to know when the ulcer crater has been completely filled in, and the patient should understand that his efforts and your efforts should be toward the healing of the disease, and this is a six-months-to-a-year program. You should discuss with him things that aggravate him and discuss with him getting full nights of rest, straightening things out on his job, getting a nap over the weekend, getting outdoors every day, and getting a regular vacation. For many years or perhaps his whole life, he should plan on a glass of milk in midmorning and midafternoon. We keep these patients in the hospital routinely for two weeks and then keep them out of work if possible one or two weeks more after which time they have then progressed from the hourly milk feedings of the first twenty-eight days of the Sippy diet and are then ready for the convalescent ulcer diet which requires a half glass of milk or milk and cream twice in the morning and twice in the afternoon at approximately an hour's interval. After hospital discharge we see the patient a two-to-four-week intervals in the office and carefully review his diet, his rest program, and hi medications. Frequently the Banthine is continued up to two to six months, and sometimes we substitute tincture of belladonna in the latter part of this period. Tincture of belladonna should be begun at approximately fifteen drops three times a day before meals and at bedtime and increased two drops per day until the patient notices some visual symptoms. This might be anywhere from twenty-five to thirty to forty drops four times daily. When the patient notices symptoms referable to his eyes, then he should decrease the dose two drops and maintain it at this level. You should reassure the patient that if he has a duodenal ulcer he has no fears of cancer, and you should also explain to him that many a greater or lesser man, including laborers or business executives, take their thermos bottles with them to work and take milk as you outline for them. A good plan is to have them take with them on trips or carry in a pocket some of the antacid tablets such as Amphojel or Gelusil which they can handily chew as a substitute for their between-meal milk feedings.

I have borrowed liberally from many sources, especially from Walter Lincoln Palmer, M.D.

Discussion

DR. HERBERT PLASS: This is a good paper. It is comforting to reflect that this conservative method of managing peptic ulcer continues to be in vogue in the best circles. However, it makes it difficult to discuss the paper because I cannot disagree with it very much. On one point I should like to add an opinion and it is that many times superficial mucosal erosions are all the evidence which can be found radiologically of ulcer in a patient with clear-cut ulcer type story extending over a period of years. This may be the matter of timing of the x-ray examination in that a real crater might be found at the site of the mucosal erosion if the picture had been taken a week or two later without treatment. Of course I am not referring to gastritis of the common variety.

A second point is that it sometimes is fun to try and predict the type of occupation of an individual who comes complaining of ulcer-like distress. I have a feeling that by and large the ulcer-bearing individual prefers to work for someone else and to report to someone else, and that he is more efficient in doing so than if he has to be the top man. This certainly is not universally true, but I think that the hypertensive type of personality is more likely to be top man, as he is also more likely to be a good salesman.

I would like to emphasize that it is not fair to the radiologist to place on his examination the burden of "ruling out" or excluding ulcer. I do not know what number or percentage of ulcers are not demonstrated by x-ray but should guess that it may be in the neighborhood of five per cent, and in the hands of less skilled radiologists perhaps it is higher than this. One certainly cannot eliminate ulcer, for instance, as a cause of gastrointestinal hemorrhage by x-ray examination. Another point which interests me is that a considerable number of patients who have ulcer, easily demonstrated by x-ray or by the presence of one of the complications of ulcer, have been quite asymptomatic over a long period of time. I believe these people come into a sort of uneasy adjustment with their ulcers, and the indigestion they suffer is so much a part of themselves that they don't regard it as a complaint. Sometimes I can find out about these by asking specifically if anti-acid medications are used or carried daily in the pocket.

I heartily subscribe to Dr. Myhre's emphasis on rest as the primary agent in treating ulcers. I am sure nothing else is more important. Finally, I should like to ask a question, "Why do so many ulcer patients interrupt treatment, discontinue their anti-acids and anti-spasmodics when they have been told to continue them, and blandly turn up a month later having the same kind of distress and not knowing what to do about it?"

DR. JOHN ROSENOW: I have been asked, as a matter of general interest for those of the audience not having direct contact with the problem of peptic ulcer, to say a few words to summarize some of the related surgical considerations.

At the outset I should like to make a distinction between gastric and duodenal ulcer. The accepted surgical procedures when done for gastric ulcer tend to be easier technically and to give better symptomatic results; the mortality is less, and there are fewer postoperative complications. The complication of anastomotic ulcer is rare following resection for gastric ulcer; this is not the case with duodenal ulcer.

The one great distinction between the two types of ulcer that must influence one's thinking in regard to gastric, as opposed to duodenal, ulcer is the question of malignant potentiality. Malignancy in duodenal ulcer is extremely rare. On the other hand, a fair summary of the experience in the literature, I think, would be to say that ten per cent of apparently benign gastric ulcers are actually malignant. I came across an interesting statistical comparison of the surgical versus the medical treatment of gastric ulcer in the new book by Moore and Harkins (Moore, H. G., Jr. and Harkins, N. H.: The Billroth I Gastric Resection. Boston: Little, Brown and Co., 1954). This contains statements that seem surprising but actually represent a reasonable summation of the figures appearing in the literature. Let us take two groups of one hundred cases each. One group will be treated medically, and the other will be treated surgically. First the surgical group—six patients will have carcinoma and six of them will die; two of the total series will die from operation, and nine patients will have disabling postgastrectomy symptoms. Thus a total of seventeen of the surgical patients will not be benefited (including eight deaths) and eighty-three will be benefited. In the medical group, all ten of the carcinoma patients will die. Twenty of the rest will have difficulty of a serious nature in the form of intractability, hemorrhage, perforation or obstruction, and about two of these twenty will die, so a total of thirty of the medical patients will not be benefited (including twelve deaths) and seventy will be benefited.

In considering the general indications for surgery in peptic ulcer, bear in mind that the following indications apply equally well to both gastric and duodenal ulcers, over and above the special consideration of malignant potentiality in those of the gastric variety. On the question of *perforation* there is pretty widespread agreement that the treatment of choice is simple closure of the perforation. However, in recent years there is a good deal of writing being done in this country advocating resection in these people; most men are hesitant to accept this. I think that perhaps in the patient who has been seen early, who is in excellent general condition, and who has had serious trouble with an ulcer for a long time in the past (and therefore might be expected to have difficulty in the future), resection should be considered.

Obstruction is another generally accepted indication for surgery, almost always resection. But I would like to interpose here a word of caution. All apparent obstruction is not true obstruction, and the patient should have the benefit of hospital care of the medical variety—thorough and with the attention to detail so ably brought out here tonight by Dr. Myhre. In many cases the obstruction will be on the basis of spasm or edema, and these will relent under proper medical care.

As regards *hemorrhage*, there is some difference of opinion, there being all gradations of opinion from those who say all massive bleeders should be operated upon at once, to those who say that practically no one should ever be operated upon for hemorrhage. A few points deserve emphasis. First, bear in mind that the indication is massive hemorrhage, not just hemorrhage. The vast majority of hemorrhages will cease under adequate medical management. The surgical literature is full of work on massive hemorrhage, and I think that the things that are being written pertain to all types and degrees of hemorrhage. The decision as to whether to operate or not must be individualized. Age enters into it, certainly. Perhaps in the older patient who cannot be kept stabilized with 500 cc. of blood every six to eight hours, surgery should be the treatment of choice. In one's thinking on this matter, it is very easy (and I have heard this line of reasoning more than once) to say—"Well, the mortality for resection is only two to three per cent." In the face of massive hemorrhage, the mortality is not two to three per cent but more likely in the neighborhood of ten to fifteen per cent.

Our fourth usually accepted indication for surgery is *intractability*. And here there come about some of the greatest discrepancies in opinions over the country. I think we should remember that surgery for peptic ulcer,

although occupying a very valuable place in our therapeutic armamentarium, is no glittering panacea. Gastric resection is, after all, when one really thinks about it, a crude, barbaric assault on the human body. And it is irrevocable. It is in this group of cases with so-called intractability that the proper, really thorough, medical treatment as outlined by Dr. Myhre can make a big difference. In the course of my training, I had a rather interesting chance to compare different ways of handling this problem.

At the hospital where I interned, a chief of one of the medical services had been associated with Dr. Sippy for years, and his thinking and influence affected the treatment of ulcer at this hospital quite strongly. The medical treatment of ulcer was extremely thorough and meticulous, and few patients came to surgery because of intractability alone. Then, when I entered upon my surgical graduate work at another institution, I had some work during that time in the medical sections. I was quite impressed in a not entirely agreeable way with the difference as to medical treatment of ulcer that was sometimes evident. The treatment of ulcer does not consist of giving the patient a diet form, chatting hastily with him as to the great importance of taking it easy, and giving him about ten prescriptions, and writing his doctor. I am sure that many patients over the country as a whole who are subjected to surgery on the basis of intractability would get by on medical treatment of the thorough type described to us tonight.

After all, there is about a five to ten per cent incidence of serious "dumping" of a disabling type after gastric resection, as well as other troubles. You'd rather not have the postgastrectomy patient coming back to you, and saying, "Ye gods, give me back my ulcer." In this connection, I think a rather forthright statement of a preceptor of mine at one stage in my training might well be quoted, namely, that for a patient to be advised to have surgery (in the absence of perforation, hemorrhage, or obstruction), he felt that the patient should have had "one h—l of a lot of trouble." My own personal feeling, had I a troublesome duodenal ulcer, without other complication, about having surgery would be about along those lines—that I'd have to have "one h—l of a lot of trouble."

DR. WALTER UDE: It has been a privilege to hear this splendid paper. The essayist is to be complimented. He has not only given us a complete analysis of the problem in hand, but has also outlined a program of management and therapy which indicates a wide personal knowledge and experience.

A careful and complete radiological examination of the upper gastrointestinal tract plays an important part in the study of patients suspected of ulcer. The fact that the radiological examination should not be the primary procedure to be placed ahead of a careful clinical study is self-evident, but is too often ignored. Certain limitations, which we cannot deny, make it impossible to always "rule in" or "rule out" an ulcer. For instance, we are limited in the extent of the examination in the presence of gross hemorrhage, or in the presence of marked debility from other causes. The case may be referred after a prolonged therapeutic management which may have considerably modified the findings. The ulcer may also be so located that it can be demonstrated only with great difficulty, often in a region where it is not accessible to palpation. A close co-ordination, therefore, of the clinician's findings and the radiologist's examination will yield far better results than the performance of the radiological examination as a routine procedure without direct guidance by the clinician. This brings us back to the age-old admonition that the request for the examination should bring with it this guiding information. Without it the radiologist attempts to get what information he can from the patient while he does the examination, a procedure which certainly cannot compete with the analysis of the clinician.

Permit me to discuss briefly difficulties encountered in four distinct regions of the upper portion of the gastrointestinal tract:

1. Hiatus hernia may be readily demonstrated in the routine examination, but at times is not so readily detected. In my experience, the shortened esophagus with hiatus hernia is best shown by studying the lower end of the esophagus in the prone oblique position during the ingestion of barium suspension through a drinking tube. This method is more successful than an attempt to demonstrate hernia in the Trendelenburg position. Ulcer in the herniated portion of the stomach may be obscured by the barium suspension, but often is clearly in evidence. It should always be kept in mind in cases of hiatus hernia.

2. The high, posterior wall of the stomach is a region where ulcer may be easily overlooked, especially in the sthenic individual.

3. Prepyloric ulcer, either on the posterior wall or on the lesser curvature, may be lost in prominent mucosal folds or obscured by peristaltic activity which may seem to obliterate a suspicious fleck of barium retention. Here, compression spot films with demonstration of mucosal pattern are of great help. If ulcers in this area fail to improve or even seem enlarged after therapeutic management they must be considered as questionably malignant, even though all other findings point toward benign ulcer.

4. Postbulbar ulcer of the duodenum is frequently the cause of silent bleeding from the upper gastrointestinal tract. Our pathologists have impressed us with the importance of finding these ulcers. Penetration directly into the pancreaticoduodenal artery may lead to fatal bleeding, and thus the pathologist will find the ulcer. The radiologist should be just as keenly interested in this region of the duodenum as he is in the duodenal cap. If there is persistent spasm and marked change in the mucosal pattern, a presumptive diagnosis of hidden postbulbar ulcer should be entertained as a working diagnosis, even though a definite crater cannot be found. Where such ulcers are of relatively short duration, the spastic contracture may completely hide the crater. Later

re-examination, often after only a few days, may visualize the crater.

We should not hesitate to re-examine a patient if there is the least doubt as to the findings, or if the clinician is not satisfied that the problem has been solved. We frequently recall a patient because of some suspicious finding on routine films, or the presence of hypersecretion or food retention in the stomach. We do not hesitate to examine in the presence of active bleeding, but avoid deep palpation or compression.

Let me again emphasize the necessity of a close co-ordination of the work of the clinician and the radiologist. Such co-operative effort yields dividends.

DR. R. S. YLVISAKER: I enjoyed Doctor Myhre's paper very much. In my opinion his excellent summary indicates to us especially the main details which go into good medical care of the peptic ulcer patient. The importance of the subject of peptic ulcer is well known to all of us, and it is also indicated by the frequency with which many phases of this subject are covered in the annual meetings of the American Gastroenterological Association. Dr. Anton Carlson once remarked at one of these meetings that the meeting of the American Gastroenterological Association might be referred to as the "Annual Symposium on Peptic Ulcer."

I believe the main point that Doctor Myhre stressed in his paper was the need for a meticulous attention to detail in both the accurate diagnosis and the treatment of peptic ulcer. One is often discouraged with the laxness with which medical treatment is prescribed in this condition. In fact, many papers have appeared in the literature, even by men well respected in their fields who recommend more frequent surgical treatment because of failure of medical management when such management has been anything but careful. There is no question in my mind but that a good ninety-five per cent of peptic ulcers can be cured or well controlled by careful medical management carried out over a protracted period of time.

I would like to comment briefly on a few other points mentioned by Doctor Myhre:

1. The well-known dictum of no acid, no ulcer. This is undoubtedly the general rule and the safest guide to follow. In regard to gastric ulcer, any ulcer that appears in the presence of achlorhydria should be considered malignant until proved otherwise. However, I am sure that all of us have had the experience of occasionally finding a benign gastric ulcer even in the presence of a well established achlorhydria.

2. In regard to the incidence of peptic ulcer in hypertension, it is true that the usual case of peptic ulcer is apt to have a low to normal blood pressure and a slow pulse. However, I have recently had several cases of proven peptic ulcer come under my care with hypertension.

3. I cannot accept the position held by some of routinely advising surgery in gastric ulcer, chiefly for two reasons. The first is that by meticulous attention to detail and calling in every possible diagnostic aid, including gastroscopy in suspicious cases, I believe that the error of overlooking malignant ulcers can be dropped down well below the ten per cent mark. In the second place, I frankly do not believe that the results of surgery in cancer of the stomach are good enough to warrant submitting ninety per cent of the people to unnecessary surgery when perhaps only one out of ten of the malignant cases can be cured. Here again, each case must be studied individually and all factors evaluated before a choice of method of treatment is made.

4. In regard to gastric hemorrhage, I believe it is a mistake to routinely transfuse these cases on admission to the hospital. There is evidence to show that immediate transfusion increases the risk of continued bleeding, recurrent bleeding, and even increases the mortality rate. By far the majority of cases of gastric hemorrhage will subside promptly on complete bed rest. The crying need is for closer observation of each case and the use of transfusion only in those in which evidences of shock persist. Later after there is good evidence that bleeding has stopped, if the hemoglobin is low, then it is justifiable to transfuse in order to shorten the period of convalescence and overcome the severe anemia.

DR. JAY C. DAVIS: I do not agree with Dr. Myhre that all patients with an ulcer should be hospitalized. There are some patients who can be healed while ambulatory. There are some who respond to treatment faster if they can get away on a vacation out of their own city and completely away from all disturbing factors that may exist in their family life or business.

Psychosomatic factors are very important and until the factors can be discovered and talked over with the patient one may not succeed in curing the patient.

Dr. Ylvisaker stated in his discussion that all peptic ulcer hemorrhages do not need surgery. It is important, however, to remember that slow bleeding over a period of many days may result in an anemia and drop in blood pressure severe enough to cause angina and changes in the electrocardiogram suggesting coronary insufficiency especially in an older patient with coronary sclerosis. A massive hemorrhage with a marked drop in blood pressure may result in such a marked coronary insufficiency that myocardial infarction may ensue without closure of a coronary artery. If the fall in blood pressure and the hemorrhage is massive, or if more than one severe hemorrhage occurs before surgery is resorted to the patient may suffer cerebral damage from anoxia. Degeneration of the kidney tubules may also occur with uremia resulting, or the patient may die in hepatic coma if a damaged liver existed at the time of the hemorrhage.

Why Milliequivalents?

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THE FOLLOWING presentation says nothing new or original. It is an attempt to answer the question posed by the title by giving in as simple a form as possible, some of the information gleaned from the literature about milliequivalents as a system of units. This information is too often found in journals not readily available, or in highly technical and complicated form.

What is a milliequivalent? The chemical potency of a substance in solution depends on the amount of it present by weight, the substance's atomic weight and the substance's valence. The comparative potency of the substances in solution can be expressed according to how much hydrogen ion they will neutralize or be equivalent to. The unit of measure in chemistry is the equivalent.

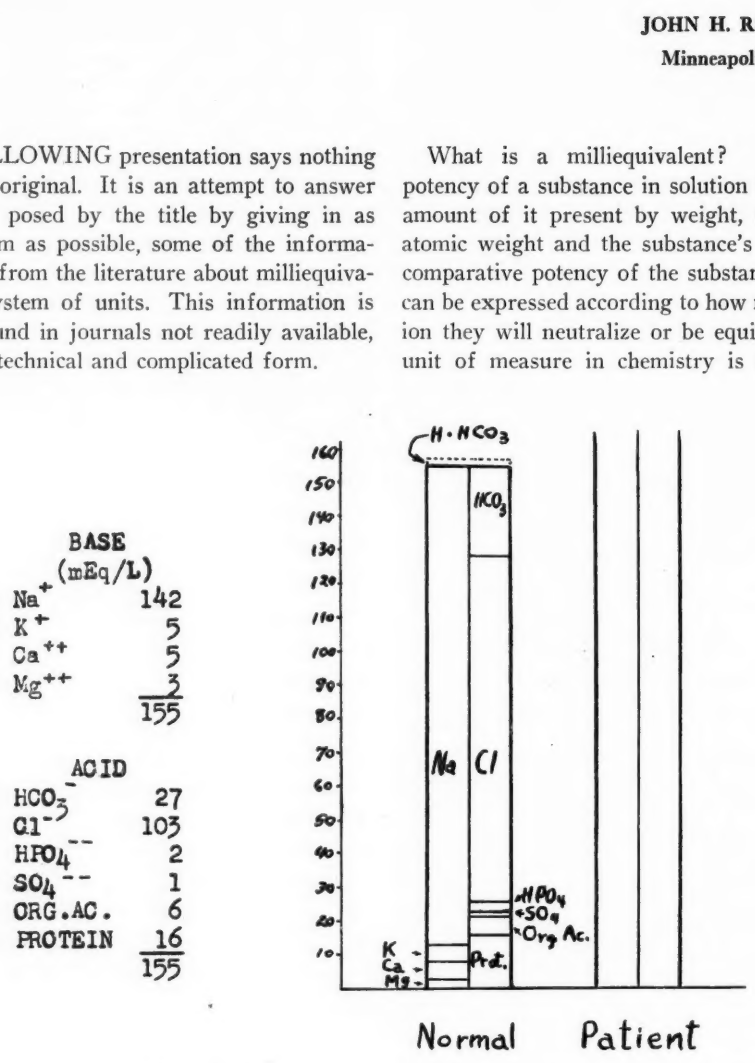

Fig. 1. Acid-base composition of blood plasma.[4]

Definitions

An electrolyte is a chemical substance, which when dissolved in a solution, separates or dissociates into electrically charged positive and negative particles, or atoms, called ions. The positive ions are called cations and the negative ions are called anions.

An equivalent is the weight of an ion, which in neutralization reactions, is equivalent to, or will neutralize one gram-atom of hydrogen. For example, one atom of chlorine will neutralize one atom of hydrogen since they are of the same valence, a valence of one. Therefore, since the weight of one atom of chlorine expressed in grams is 35.5, and that of hydrogen is one, then 35.5 grams of chlorine will neutralize or be equivalent

Abridgement of inaugural thesis presented before the Minneapolis Academy of Medicine, March 16, 1953.

to one gram of hydrogen. If the amount of chlorine is double, then we have two equivalents of chlorine present, and so on. Again, for example, a single atom of calcium, having a valence of two, will combine with or neutralize two atoms of chlorine. Since the atomic weight of calcium is 40 and that of chlorine is 35.5, then 40 grams of calcium are equivalent to twice 35.5 or 71 grams of chlorine in a chemical solution. Equivalent=(grams x valence)÷atomic weight. Equivalents as units of measure are satisfactory for use in chemistry but not in medicine. In medicine, the amounts of ion dealt with are too small, so the milliequivalent is used, or one thousandth of an equivalent. Milliequivalents are expressed as units per liter, and are abbreviated "mEq" or "meq." Under normal conditions, there are 155 mEq of basic ions (cations), and 155 mEq of acid ions (anions) per liter of plasma. For practical purposes, the sum of the basic ions always equals the sum of the acid ions.

General Remarks

It would seem high time that we stop considering the various electrolytes as so much dead weight measured in ounces on the grocer's scales. We are dealing, in plasma, not with amounts by weight of chemical salts such as NaCl, KCl, and the like, but with separately controlled, independently reacting ions such as Na, K, Cl, and so on. The ions are active elements and we should evaluate them on their chemical activity. To do this and to understand the actual chemistry of fluid balance is to talk in terms of mEqs. It would be folly, for instance, to compare engines solely on the basis of their weight. A more valid and useful comparison is on the basis of their capacity for producing power, i.e., horsepower. One gram of chloride ions in plasma is roughly 100 times as powerful as one gram of protein when measured from the standpoint of chemical combining capacity, and 800 times as powerful osmotically. One gram of sodium in plasma is about 150 times as potent chemically and 1,200 times as potent osmotically as one gram of protein.

The rating of ions in the fluids of the body on the basis of such differing units as milligrams per 100 cc., grams per 100 cc., and volumes per cent, is perhaps like trying to work with fractions having different denominators. It would be impossible to add ⅓, ¼, and ⅛, let us say, if we did not have that very useful echo from grade-school days, the least common denominator. It then becomes simple to add 8/24, 6/24, and 3/24 to get the answer of 17/24. MEqs might be thought of as the least common denominators of fluid balance.

An example of this and of the differing worth of dead weight units and activity units (or mEqs) is readily found in a patient who loses 7.0 grams of chloride and 4.6 grams of sodium in tube drainage. It would be immediately apparent only to a chemist that loss of basic and acid ions is going on equally. If it were stated, as is actually the case, in this instance, that the patient lost 200 mEq each of chloride and sodium, the acid-base significance would be clear. It would also be clear that the relation of the two is high in chloride relative to extracellular fluid composition where the ratio is 142 of sodium to 103 of chloride. He will deplete his chlorides faster than his sodium. The loss of such a solution wholly from extracellular sources would thus lower chloride concentration differentially and produce metabolic acidosis despite the chemically neutral nature of the loss.

Examples of Use of Milliequivalents

Before embarking on the examples, let me call attention to the normal values in Figure 1. The first example is based on the fact that the sum of the basic ions equals the sum of the acid ions. Before the development of the flame photometric method of analysis of plasma, the only way to arrive at a rapid, rough estimate of the electrolyte concentrations, especially that of sodium, was by application of the equality of the acid and base ions, using other more easily obtainable values, such as for HCO_3 and Cl. And where a flame photometer is not available it still can serve, in spite of obvious shortcomings. For it to be valid, we must be able to assume that potassium is normal, and that the acid ions other than HCO_3 and Cl are present in normal amounts, often, of course, not the case.

(R^+ represents the sum of the basic ions other than sodium, and R^- represents the sum of the acid ions other than HCO_3 and Cl.)

sum of base ions	=	sum of acid ions
Na + R^+	=	HCO_3 + Cl + R^-
Na + 13	=	HCO_3 + Cl + 25

Now let us say that we have values for HCO_3 and Cl of 23 and 88 mEq/L respectively.

$$\begin{aligned} Na + 13 &= 23 + 88 + 25 \\ Na &= (23 + 88 + 25) - 13 \\ Na &= 136 - 13 = 123 \text{ mEq/L} \end{aligned}$$

The value for serum protein in mEq could of course be inserted or interpolated in the equation and would increase its accuracy somewhat. This method, while a thin reed to lean on, can be used if the exceptions and inaccuracies are kept in mind, with an occasional check by the older, time-consuming direct chemical determination of sodium.

Let's take up another oversimplified example. One could estimate roughly the amount of solution necessary to return the electrolyte concentration to normal. If we assume that the extracellular fluid volume is 20 per cent of the body weight (or body weight÷5), the following formula could be used as an approximate guide:

$$\text{Total amount of ion needed} = \frac{\text{Pt's wt. in kg.}}{5} \times \left\{ \begin{matrix} \text{normal value} \\ \text{of ion in} \\ \text{mEq/L} \end{matrix} - \begin{matrix} \text{pt's level} \\ \text{of ion in} \\ \text{mEq/L} \end{matrix} \right\}$$

Let's run through that with an example of a man weighing 175 pounds, who has a sodium of 132 mEq/L, remembering, of course, that to convert pounds to kilograms, divide by 2.2. His weight is thus 175÷2.2=80 kg.

$$\text{Total needed} = \frac{80 \times (142-132)}{5} = 16 \times 10 = 160 \text{ mEq}$$

Physiological saline contains 154 mEq of sodium per liter, so one liter would give the approximate amount, theoretically, which would restore the sodium to normal.

Now for a more complicated example. Given a patient who has lost 3 liters of body fluids by suction drainage. Her normal weight is 114½ pounds (52 kilograms). She has the following values for electrolytes, which are converted using the factors found in Table I,

CO_3.....	50.6 vol.%	x 0.45 =	23 mEq/L
Cl (as NaCl)	550.0 mgm. per 100 cc.	x 0.171 =	94 „
Protein.....	7.4 Gm. per 100 cc.	x 2.43 =	18 „
Sodium .....298.0 mgm. per 100 cc.		x 0.435 =	130 „

The patient's three liter loss of fluid, for the purposes of this example, is presumed to come from the extracellular fluids. So we first figure the normal amount of extracellular fluid for this patient and then subtract the loss,

52 kg÷5=10.4 liters, her normal extracellular fluid
— 3.0 liters, loss

7.4 liters, extracellular fluid after loss (a rather broad assumption)

Na Normal	10.4 x 142 =	1477	mEq of Na[1]
After loss	7.4 x 130 =	— 962	„ „ „[2]
		515	„ „ „[3]

[1]Total amount normally present
[2]Amount present after loss of 3 liters
[3]Of Na which have been lost

Cl Normal	10.4 x 103 =	1071	mEq of Cl[1]
After loss	7.4 x 94 =	— 695	„ „ „[2]
		376	„ „ „[3]

[1]Total amount normally present
[2]Amount present after loss of 3 liters
[3]Of Cl which have been lost

It remains to pick the repair solutions, which can be done without too much difficulty when we know the mEq content of the various available solutions (Table II).

As to chlorides—we see from the table that normal saline has 154 mEq/L of chloride, so to supply the deficit of 376 mEq, we need 376÷154 =2.4+liters. This amount of saline will supply us with the same amount of sodium, or 376 mEq. So we are left with 515—376=139 mEq of sodium to supply in some other way. Preferably we would like a solution having the sodium in combination with an acid ion that is easily metabolized, in order not to increase unduly the acid ions. Sixth molar sodium lactate is an excellent solution for this purpose. How much is needed? A liter of this solution, according to the table, contains 167 mEq of sodium, so we need 139÷167=832 cc. of sixth Molar sodium lactate.

For practical purposes, then, we would need about 2½ liters of saline and 800 cc. of sixth Molar sodium lactate. This would give us a little more total volume of fluid than we need. These calculations are for replacement of an existing immediate deficiency and do not provide for replacement of basic requirements such as losses through lungs, skin, obligatory volume needed by the kidneys and the like. The extra fluid we gave in our example could be reckoned in when those basic requirements are being calculated.

I cannot emphasize too strongly that the fore-

going examples are purely for the purposes of illustration. They are not in any way advanced as specific guides in solving fluid-electrolyte problems. There are no pat, fool-proof rigid formulae that can be applied to all cases. But with the use of milliequivalents, at least such problems can be approached with some degree of intelligent understanding, and they may well lose some of their weird, cabalistic, mysterious aspects, and may become, while not always exactly simple, at least possible of rational solution.

TABLE I. NORMAL ELECTROLYTE VALUES—CONVERSION FACTORS

	Normal (mgm/100 ml)	×	Factor	=	Normal (mEq/L)
Na^+	317—340		0.435		137—148
K^+	16—21		0.257		4.1—5.4
Ca^{++}	9—11		0.5		4.5—5.5
Mg^{++}	1.2—2.4		0.82		1—2
HCO_3^- (CO_2 vol. %)	55—75	(or ÷2.22)	0.45		25—34
Cl^- (as NaCl)	585—645		0.171		100—110
(as Cl)	352—389		0.282		100—110
PO_4^{--}	3—5		0.58		1.7—2.9
SO_4^{--}	1.6—2.4		0.625		1.0—1.5
Protein (Gm. %)	6.5—8.0		2.43		16—19

$$\text{Concentration (mEq/L)} = \frac{\text{Concentration (mgm/100 ml)} \times 10}{\text{atomic weight}} \times \text{Valence}$$

$$\text{Conversion factor*} = \frac{10}{\text{atomic weight}} \times \text{Valence}$$

*does not apply to HCO_3, PO_4, and protein.

TABLE II. ELECTROLYTE CONTENT OF INFUSION FLUIDS (mEq/L)

Fluid	Na	K	Ca	Cl	Effective CO_2	NH_3	Mg	HPO_4
NaCl 0.9%	154.0			154.0				
Sodium bicarbonate 1.2%	143.0				143.0			
Ammonium chloride 0.75%				140.0		140.0		
KCl ampules (1.59 Gm.)		20.0		20.0				
Ringer's solution	147.0	4.0	4.5	155.5				
Butler's solution	30.7	20.8		23.2	20.5			7.8
Hartmann's solution	136.0	5.3	3.6	112.0	33.0			
Aminosol	6.1	0.053		17.0				
Amigen	30.0	15.0		42.0				
Travamine	110.0	5.6		65.0				
Plasma (250 cc.)	35.5	1.25	1.25	25.75	6.75		0.75	0.5
Whole blood (500 cc.)	40.0	2.0		16.0				
Polysal (Cutter)	140.0	10.0	5.0	103.0	55.0		3.0	
Mod. duod. solution (Baxter)	80.0	36.0	4.6	63.0	60.0		2.8	
Travert 10%—Electrolyte Solution No. 1 (Baxter)	80.0	36.0	4.6	63.0	60.0		2.8	
Travert 10%—Electrolyte Solution No. 2 (Baxter)	57.0	25.0		50.0	25.0		6.0	12.5
Travert 10%—Electrolyte Solution No. 3 (Baxter)	63.0	17.5		150.5		70.0		
Ammonium chloride 2.14% (Baxter)				400.0		400.0		
Darrow's solution (Baxter)	121.0	35.0		103.0	53.0			
M/6 sod. r-lactate (Baxter)	167.0				167.0			
Travert 10%—KCl 0.3% in water (Baxter)		40.0		40.0				
Travert 10%—KCl 0.3% in 0.45% NaCl (Baxter)	77.0	40.0		117.0				

Discussion

In the field of fluid-electrolyte balance, a vast amount of research is being done, and major advances and changes in concepts are taking place. The work is, and of necessity must be, done in terms of milliequivalents. The article in the literature on this subject which reports in other terms than milliequivalents is rare indeed. If we wish to have an intelligent understanding of what is being done, and want to put it to enlightened practical use, we should be familiar with the language in which it is written, so to speak.

For those of us who do not work exclusively in the so-called teaching institutions, the habit of thinking and working in terms of milliequivalents is made difficult to acquire by the fact that most hospital laboratories report electrolytes in the older system. It is suggested that the advent of the newer system might be hastened if hospital laboratories were asked routinely to report all electrolyte values in both systems for a time, with the eventual end in view of reporting only in milliequivalents. The conversions can be done far more easily in the laboratory, from prepared tables and graphs, without the necessity for actual calculations. Thought might be given to the provision in charts of a special electrolyte form, either separately or as part of the regular laboratory sheet; part could be a form for the reporting of the patient's values in both systems, and part could be a table of normal milliequivalent values.

It is unquestioned that in the ordinary case, all of this isn't too necessary. But the occasional complicated problem almost demands use of the milliequivalent system for intelligent handling.

Summary

It is hoped that the foregoing presentation may in some small measure help to answer the question posed by the title by bringing together from the literature some items of interest, edification, illustration and justification regarding the use of milliequivalents.

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REGULAR CORPS EXAMINATION FOR MEDICAL OFFICERS

A competitive examination for appointment of Medical Officers to the Regular Corps of the United States Public Health Service will be held in various places throughout the country on June 7, 8, and 9, 1955.

Appointments provide opportunities for career service in clinical medicine, research, and public health. They will be made in the ranks of Assistant and Senior Assistant, equivalent to Navy ranks of Lieutenant (j.g.) and Lieutenant, respectively.

Entrance pay for an Assistant Surgeon with dependents is $6,017 per annum; for Senior Assistant Surgeon with dependents, $6,918. Provisions are made for promotions at regular intervals.

Benefits include periodic pay increases, thirty days' annual leave, sick leave, medical care, disability retirement pay, retirement pay which is three-fourths of annual basic pay at time of retirement, and other privileges.

Active duty as a Public Health Service officer fulfills the obligation of Selective Service.

Requirements for both ranks are U. S. citizenship, age of at least twenty-one years, and graduation from a recognized school of medicine. For the rank of Assistant Surgeon, at least seven years of collegiate and professional training and appropriate experience are needed, and, for Senior Assistant Surgeon, at least ten years of collegiate and professional training and appropriate experience are needed.

Entrance examinations will include an oral interview, physical examination, and comprehensive objective examinations in the professional field.

Application forms may be obtained by writing to the Chief, Division of Personnel, Public Health Service, Department of Health, Education, and Welfare, Washington 25, D. C. Completed application forms must be received in the Division of Personnel no later than May 6, 1955.

Clinical Aspects of Fluid and Electrolyte Management

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IN THE PRACTICAL day-to-day management of a patient on total, or even partial, artificial alimentation it would be ideal if one could order a uniform or standard intravenous feeding solution. However, in illness and stress every patient presents a variation that must be accurately dealt with.

Fluid, electrolyte, nitrogen and caloric needs require an assessment of basic needs, an estimate of deficits to be repeated and accurate information on losses that are occurring.

Adequate caloric and nitrogen intake is difficult to achieve by intravenous solutions unless all of the nutritional elements are utilized, and therefore the basic solution should contain 5 or 6 per cent amino acids to provide nitrogen in 10 to 15 per cent invert sugar or fructose to provide the carbohydrates, and 5 to 7.5 per cent alcohol to provide the additional calories which are ordinarily supplied by fat. Such a solution contains 1000 calories per liter and is capable of supplying at least 100 per cent of energy requirement without exceeding the fluid needs. All the fluid requirement of any but an anuric patient should be supplied in this form.

In the sick individual the fluid and electrolyte needs may vary greatly from the average normal. There is, therefore, no substitute for frequent, detailed analyses of the chemical levels and of careful calculation of balances determined from body fluid and excretion levels. With this information one can then more accurately order the volume of fluid and quantity of electrolytes as they are needed from day to day. The basic solution of amino acids and invert sugar or fructose must be one of low sodium, potassium and chloride content so that calculated quantities may be added to accommodate the requirement of the patient.

Such electrolyte additions may be easily made by injecting them into the basic solution from stock concentrates made up in sterile vials by a pharmacist. In this manner the physician can avoid ordering a liter of this and a liter of that—a method certain to be deficient in one or more aspects of nutrition.

At the outset a base line must be determined. Weight, hemoglobin, hematocrit, carbon dioxide, chloride, BUN, protein, urine specific gravity, and usually serum sodium and potassium are sufficient. The state of hydration and deviations from electrolyte equilibrium are then evident. If a deficit is present it is obvious that the balances attained on succeeding days must be on the positive side until repletion is achieved.

Fluid balance can be maintained only by careful attention to the following.

1. An accurate intake and output record must be kept. In this regard meticulous endeavor by the nursing staff is indispensable. An inlying catheter may be required in seriously ill or incontinent patients. Nasal suction should always be collected into a trap bottle.

2. Calculation or estimation of insensible water loss. The Darrow formula (adult) of 44 cc. insensible water loss for each 100 calories metabolized is quite easily calculated from the DuBois chart of the Lilly Co. slide rule for diabetic diets. Regardless of the caloric intake, an individual must satisfy his energy demands. From height, age, weight and sex factors on the DuBois scale one can find the caloric requirement. For normal bed activity and specific dynamic action the factor of basal calories plus 40 per cent is employed. To this factor 8 per cent is added for each degree of

Supported in part by a grant from Baxter Laboratories, Morton Grove, Illinois. Robert Herwick, M.D., Medical Director.

Presented by title as thesis before the Minneapolis Academy of Medicine, March 16, 1953.

fever. The wide variance in insensible fluid loss is demonstrated by the following examples.

(A) A 6′, 200-pound man, thirty-five years old, with temperature of 102.6°

Basal	= 2000 calories
Bed activity) + 40% and SDA)	= 800 calories
3° fever × 8 =) + 24% ¼ of 2000)	= 500 calories
	3300 calories requirement

33 × 44 = 1452 cc. insensible water loss per 24 hours.

(B) 5′ 2″ 100 pound woman, thirty-five years of age, with normal temperature

Basal	= 1240 calories
Bed activity) + 40% + SDA)	= 500 calories

17.4 × 44 = 766 cc. insensible water loss.

From these examples one can see at a glance that reliance upon estimate of, say, 800 to 1000 cc. insensible water loss would lead to serious aberration in fluid balance before many days had passed.

3. Daily body weight, when determined carefully with proper apparatus for bed patients, is an ideal method of following fluid balance. Before dehydration becomes clinically evident a 6 per cent loss of body weight has occurred (9 pounds in a 150-pound man). When the measured fluid output and calculated insensible water loss are subtracted from fluid intake and this figure checked against body weight, one has a fairly accurate evaluation of a patient's fluid status.

Electrolyte Balance.—Reliance upon a normal or average behavior of electrolyte physiology may lead to disaster. Laboratory determinations of serum levels and excretion quantities provide a far greater safety.

1. The state of hydration has a profound effect upon the electrolyte status for overhydration leads to dilution of electrolytes and dehydration causes serum electrolytes to rise above normal levels.

Since the symptoms of electrolyte disturbances are direct results of the serum concentration levels rather than total body quantities, it is important to realize that an untreated patient who has suffered loss of both water and electrolytes may, under certain circumstances, have an entirely normal serum electrolyte pattern and therefore no clinical symptoms of electrolyte deficit. It is such a patient who, on repletion of too much water in proportion to electrolytes, will surprise the physician when he develops severe symptoms of mineral deficit. Conversely, a patient who has been overtreated (usually by saline solution) may exhibit symptoms of hyperconcentration if, in recovery of edema, he loses water faster than electrolytes.

TABLE I. BLOOD ELECTROLYTES IN PATIENT DEVELOPING HYPOCHLOREMIC SHOCK AFTER HYDRATION

	Adm.	4th Day	4th Day +3 Hr.	5th Day	11th Day
Hematocrit	56	46		47	36
CO_2 comb. power mEq/L	31	46	30	31	27
Chloride mEq/L	97	71	103	86	98
BUN	15				72
Sodium mEq/L	149				156
Potassium mEq/L	3.8				2.3
Cumulative Na bal. mEq.		+23			+739
Cumulative K bal. mEq.		+2.1			+211
Cumulative Cl bal. mEq.		+49			+736
Cumulative Fluid bal. cc.		+804			+2449
		Day of shock			

TABLE II.

	Number of Determinations	Volume cc	Average mEq/L		
			Sodium	Potassium	Chloride
Gastric suction* No oral intake	68	795	64	9.7	87
Gastric suction* Oral intake av. 792 cc.	108	1267	45	6.1	89
Gastric suction after gastrectomy. Oral intake average 269 cc.	86	603	70	7.5	63
Urine	518	1404	64	36.0	39
Bile	51	195	136	4.7	100

*Gastrectomy patients not included.

Thus, one must gather as many facts as possible on the state of hydration in order to properly evaluate the significance of serum electrolyte determinations.

To illustrate, let us assume a hypothetical case of dehydration and consider the chloride ion:

Admission: Hematocrit, 56, chloride 97 mEq, weight 50 kg.
Normal: Hematocrit, 40, chloride 104 mEq

TABLE III. TABLE OF AVERAGE URINE EXCRETION RATIO OF SODIUM TO CHLORIDE

300 Determinations mEq/L			
	Pre-op.	Post-op.	Total
Sodium	90.8 mEq (5.3 gm.)	37.0 mEq (2.2 gm.)	62.6 mEq (3.7 gm.)
Chloride	86.7 mEq (5.1 gm.)	44.3 mEq (2.6 gm.)	64.1 mEq (3.8 gm.)

water are divided by 3, thus, 117 mEq cl. and 1333 cc. of water are added to each day's chloride and fluid requirement for three days.

TABLE IV. EXAMPLES OF DEVIATIONS FROM THE AVERAGE

	Na:Cl Urine Excretion Ratio. Daily Determinations (mEq/L)							
Case No. 1	Sodium	252	116	140	2?0	324	247	
	Chloride	118	49	113	62	65	26	
Case No. 2	Sodium	132	148	185	99			
	Chloride	19	10	3	2			
Case No. 3	Sodium	125	91	2	5	5	5	2
	Chloride	71	79	18	38	48	38	70

TABLE V. RELATION OF SERUM POTASSIUM TO BALANCE mEq/L

Case No. 1	Serum K	4.9	2.9		2.7		3.0		
	Daily K Bal.	−3.1	−114.6	−51.0	−26.0	+5.9			
Case No. 2	Serum K		3.2			2.4		5.2	
	Daily K Bal.	−41.0	−15.0	−11.0	−29.0	+29.0	+199.0		
Case No. 3	Serum K	5.8					2.9		4.0
	Daily K Bal.		−41.0	−39.0	−3.0	−27.0	−89.0	+124	+56

Since the chloride level is determined from extracellular fluid space, and since this space is 20 per cent of total body, take 20 per cent of 50. = 10 liters (this patient's normal extracellular fluid volume).

Hematocrit of 56 — 40 = 16. 16/40 = 40 per cent less fluid than normal.
40 per cent × 10 liters = 4 liters deficit.

To calculate predicted serum levels in re-hydration, a proportion may be set up.

Pt. hemat.:pt. cl.::normal hemat.:expected cl.
56:97::40:cl′
56 cl′ = 3880
cl′ = 69 mEq/L

Thus 69 mEq/L would be the chloride level in the serum if the 4-liter water deficit were replaced without giving any chloride ion.

To calculate the total existing chloride deficit in a 10-liter space (pt. normal):

69 × 10 = 690 mEq cl actually present on admission.
104 × 10 1040 mEq cl normal chloride content of 10 L. space.
1040 — 690 = 350 mEq cl calculated total deficit of chloride in extracellular space.

In order to adjust to shifts that may occur and to permit re-evaluation in the course of repletion, a wise plan is to replete the calculated deficit over a three-day period. Therefore, in this example, the total deficit of 350 mEq cl. and 4 liters of

The serious drop in serum electrolyte concentration that follows rehydration without calculating the anticipated dilution and without adequate electrolyte administration to prevent dilution may be exemplified by data from an actual case.

This patient had partial pyloric obstruction for two months. Her general condition was quite satisfactory and dehydration was not apparent clinically. Fluid administration was, in the main, without electrolytes. On the fourth day profound shock appeared. The blood pressure was near zero until Levophed (nor-epinephrine) was given. Such shock has been called "hypochloremic shock" but may well be a more general condition of hypo-osmolarity.

When shock state was discovered, the patient was given 3150 cc. of fluid containing 642 meq. sodium, 656 meq. chloride, 23 meq. ammonia, 33 meq. potassium and 33 meq. phosphate, and after the first liter, Levophed was no longer needed. In such a critical case the usual rule of cautious repletion over a three-day period was deferred and a 3 per cent concentration of sodium chloride was used in the first 500 cc. It may be noted that a normal chloride level was attained in three hours. This case also exemplifies the rise in BUN in the presence of severe electrolyte imbalance.

This elevation may occur as it did in this case in spite of a satisfactory urine volume.

It should be noted that the serum potassium generally available and is reasonably helpful if anemia or blood loss do not interfere with accuracy.

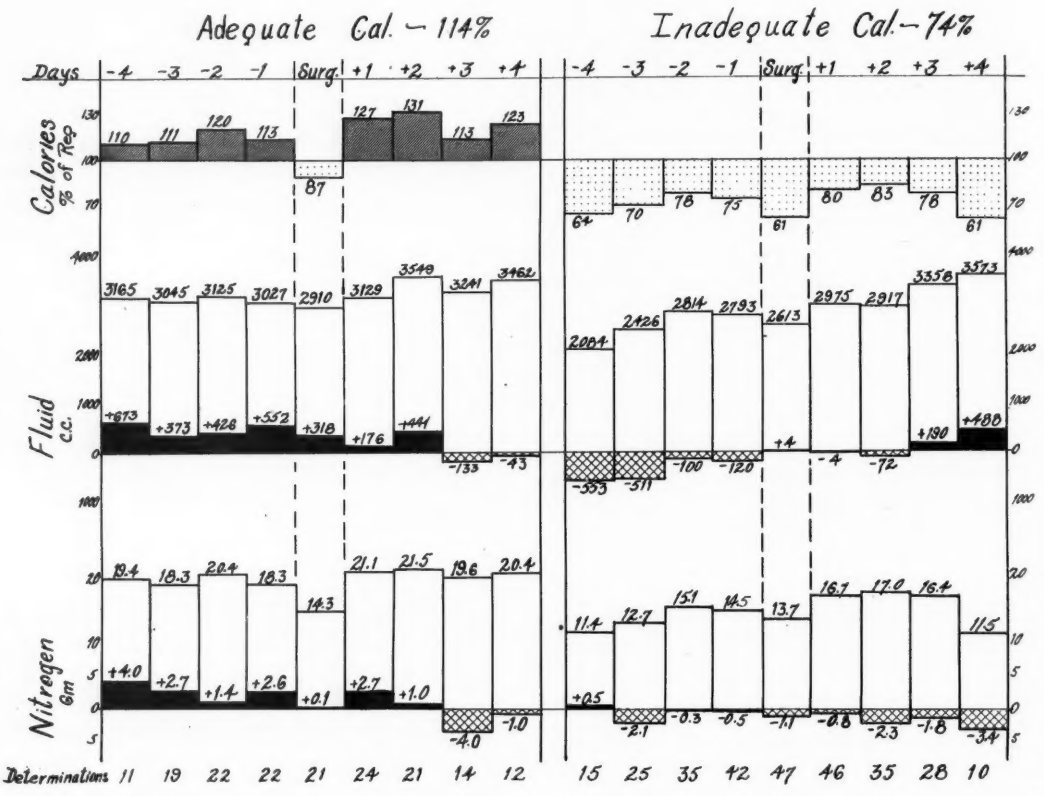

Fig. 1. Intake from zero upward; output from intake line downward. Balance indicated by +black and —cross hatch. Balance studies on 102 patients averaging five days study for each patient. Comparison between the adequate and the inadequate groups reveal a more positive nitrogen balance associated with a better caloric intake. In both groups the catabolic effect of surgery is observed in the postoperative period.

dropped to an even lower level in spite of a positive balance.

The strongly positive sodium balance no doubt is a result of giving too much sodium chloride to correct what should have been mainly a chloride deficit (pyloric obstruction). A hypernatremia of 156 mEq/L approaches a serious level. More ammonium chloride perhaps should have been given, but the ammonia ion is extremely toxic if not immediately converted to urea by the liver. If this concern is justified, then in cases of shock a rapid and safe means of pure chloride administration does not exist.

Blood and extracellular space measurements are more accurately determined by Evans Blue and inulin methods, but the hematocrit is more

Only the quantity of extracellular ions such as sodium, chloride, etc., are amenable to calculation by this method. Potassium deficit must be estimated by other means.

2. Electrolyte losses that occur from day to day in the hospital must be carefully collected. Urine, gastric suction and bile may, depending upon case urgency, be pooled into one to three day output collections.

Although rather extreme variations occur in gastric suction and particularly in urine electrolyte content, average values are of some use as a starting point.

From several hundred determinations we have compiled the following chart on average electro-

lyte concentrations in various body fluids and excreta.

In the average surgical patient sodium is excreted in an amount equivalent to the chloride excretion. The simplicity of urinary chloride determinations makes the method quite attractive. However, many physiologic influences may produce extreme variations in any given patient. Therefore, in dealing with an individual case it is unsafe to presume a 1:1 ratio between chloride and sodium. True sodium and chloride determinations should be obtained from the specimen.

The average surgical patient has a urine sodium to chloride ratio of 1:1.

The marked drop in sodium and chloride output in the postoperative period should be noted and taken into consideration in postoperative electrolyte administration.

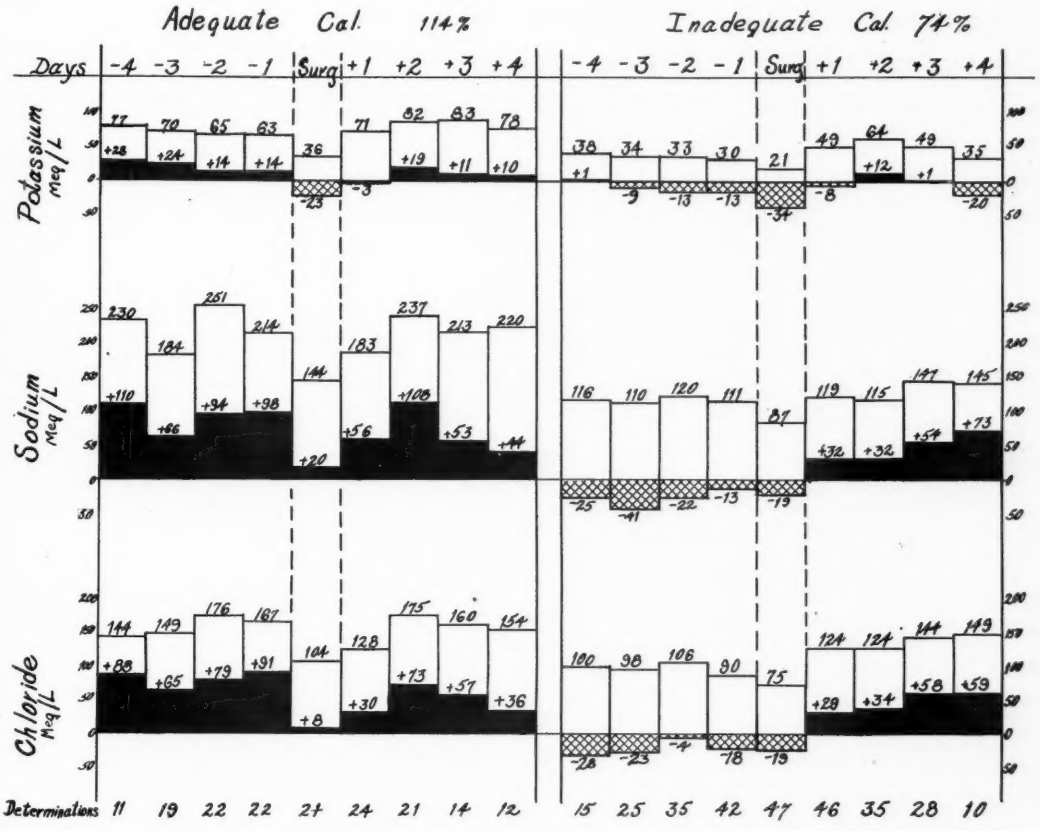

Fig. 2. Intake from zero upward; output from intake line downward. Balance is shown by +black and —cross hatch. Electrolyte balance studies upon the same group of patients in Figure 1. The potassium shift toward negativity from the preoperative to the postoperative period is not as great in the adequate calorie group. The sodium and the chloride ions exhibit an obvious difference between the adequate and the inadequate calorie groups. The intake of these ions does not vary sufficiently within each calorie group, i.e., between pre- and postoperative phase, to explain the variation in pre- and postoperative behavior between adequate and inadequate calorie groups.

Marked deviations from the average 1:1 excretion ratio frequently occur both from one patient to another and from day to day in the same patient.

3. Balance data are particularly valuable in regard to the potassium ion. The main concern in surgical patients is directed toward prevention of hypopotassemia and toward correction if it has already occurred.

Since less than 2 per cent of total body potassium is located in the extracellular fluid, a relatively large depletion (up to 50 per cent) of body

potassium occurs before the loss is reflected in the serum potassium level. Although clinical symptoms attributed specifically to a low potassium occur only after the serum level dropped at least one milliequivalent below normal, there are

Sodium balance + 800 meq.
Serum sodium rose from 158 to 170 meq/L
1/5 (extracellular fluid) × 65 (body wt. in kg.) = 13 liters extracellular fluid.
170 — 158 = 12 meq/L increase of sodium
12 × 13 = 156 meq. in extracellular space.
800 — 156 = 644 meq. of extra sodium located elsewhere than the extracellular fluid space.

CHART I. SHEET FOR RECORDING DATA

Name		Age	Wt.	Diagnosis:					
Date									
Fluid cc.	In	Oral							
		Parenteral							
	Out	Urine							
		Suction							
		Insensible							
		Other							
	Fluid Balance								
Calories		Given							
		Requirement							
	Caloric Balance								
Sodium mEq		In							
		Out							
	Sodium Balance								
Potassium mEq		In							
		Out							
	Potassium Balance								
Chloride mEq		In							
		Out							
	Chloride Balance								
Urine		Spec. Gr.							
		Sugar Gm.							
Blood	Hemoglobin (15) Gm.								
	Hematocrit (37-45) %								
	CO_2 (26-28) mEq/L								
	Cl (100-106) mEq/L								
	K (3.8-5.2) mEq/L								
	NA (137-147) mEq/L								
	Sugar (80-120) mg.%								
	BUN (8-20)								
	Protein A/G (6.7-8.4)								

many undesirable changes of cellular physiology aready under way. The presence of a strongly negative potassium balance permits a one-day to three-day prediction of drop in serum potassium level.

4. Shifts of electrolytes within the body may be detected by studies of the balance data.

For example, a patient who had received an excess of sodium exhibited the following balance findings in a combined three-day study.

Caloric Balance.—An adequate caloric intake appears to have an influence upon the equilibrium of electrolytes as well as nitrogen. It is reasonable to assume that if a patient's weight is maintained while on a sub-caloric diet that a gradual dilution of his system, a water logging, must occur since without meeting caloric requirements tissue loss is obligatory.

Complete balance studies of calories, nitrogen, sodium, potassium, chloride and fluid were recorded on 102 patients, an average of five days for

each patient. After correction for urinary loss of calories (sugar), the 102 patients were divided into two groups. One group received 100 per cent or more of caloric requirement and the other group was given less calories. Significance of this material must await completion of statistical evaluation now in progress. However, certain differences between the adequate and the inadequate calorie group are demonstrable in graphic form (Fig. 1).

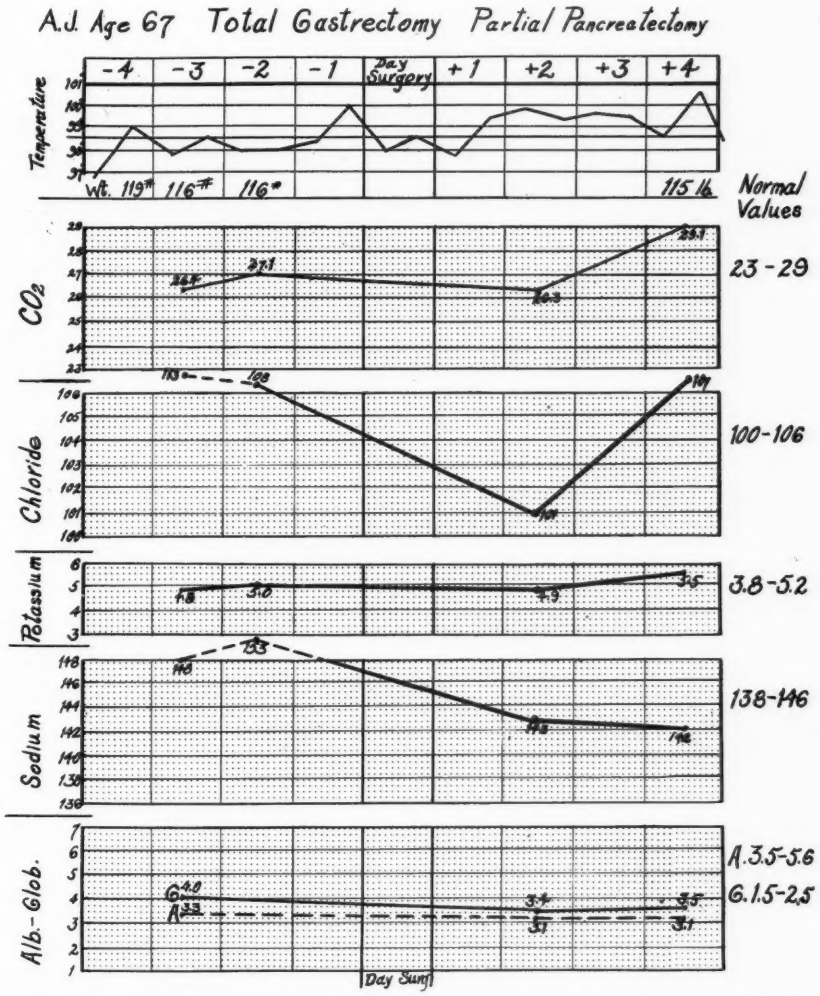

Fig. 3. Blood chemistry studies on a patient operated upon for extensive gastric cancer. Caloric intake averaged 89 per cent of requirement.

An obvious difference in the behavior of the sodium and the chloride ion exists. Even though the intake is not the same in the adequate as in the inadequate calorie group, there is noted a reversal of the preoperative and postoperative behavior from one group as compared to the other. It is doubtful that such a pattern could be explained on the basis of intake differences.

Aside from the expected postoperative excretion increase in potassium we note a proportionately smaller postoperative increase in the inadequate calorie group. One might speculate that a negative caloric balance implies greater cell breakdown to meet obligatory energy demands, thereby releasing more endogenous potassium and thus less need from exogenous sources, hence propor-

tionately greater urinary loss of administered potassium. Statistical evaluation and more accurate conclusions will be the subject of future publications on these data.

In order to achieve equilibrium consistently all possible protein as blood or amino acids must be given. In our opinion all fluid given should contain amino acids and high caloric support.

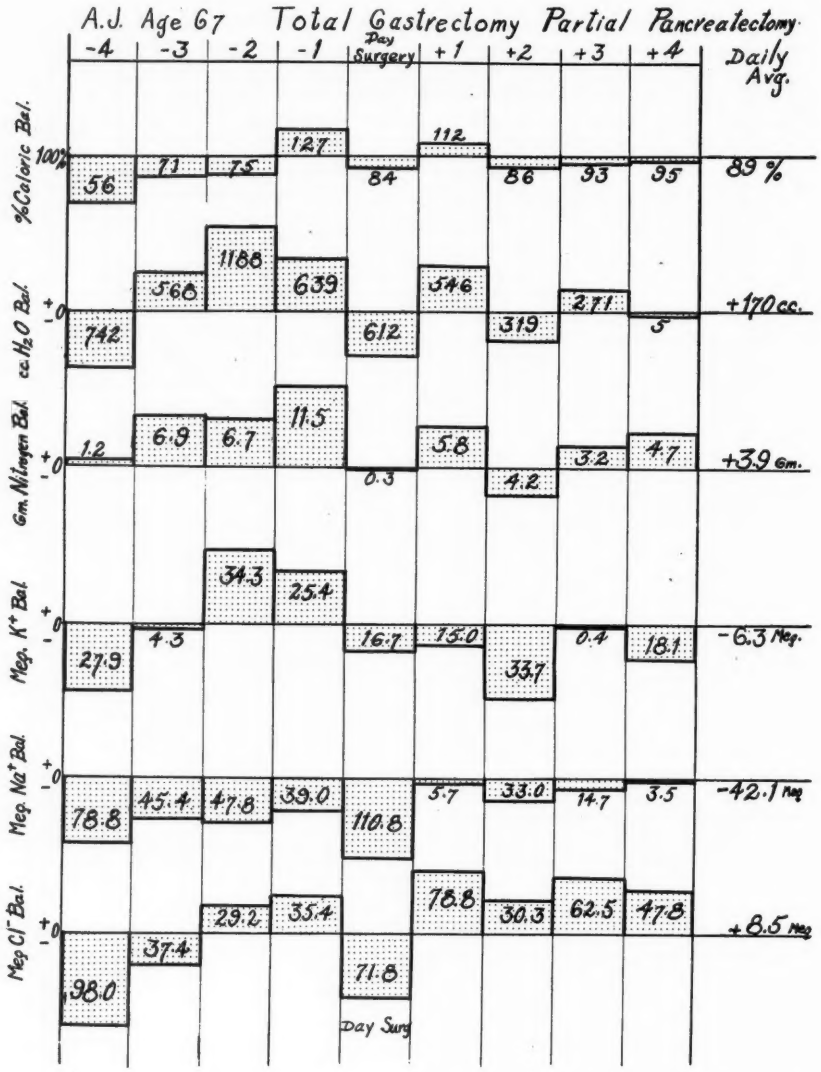

Fig. 4. Balance studies on a patient with gastric carcinoma (same as Fig. 3) reveal all balances close to equilibrium for nine days. The exception of sodium was intentional due to high initial sodium level.

Nitrogen Balance need not be calculated for the practical management of a case. Equilibrium or positive balance is of vital importance and is dependent upon adequate caloric and nitrogen intake.

Case Studies by Graphic Representation.—Figures 3 and 4 represent the blood chemistry studies and the balance calculations on a patient with carcinoma of the stomach. The moderately nega-

tive sodium balance was the result of planned therapy since the serum sodium was high at the outset. Hypernatremia is likely to be particularly bad in the face of a reversed albumin globulin requiring grafting. On a high caloric intake and close electrolyte and fluid adjustment the patient was discharged at 106 days without dressings and returned to school two weeks later.

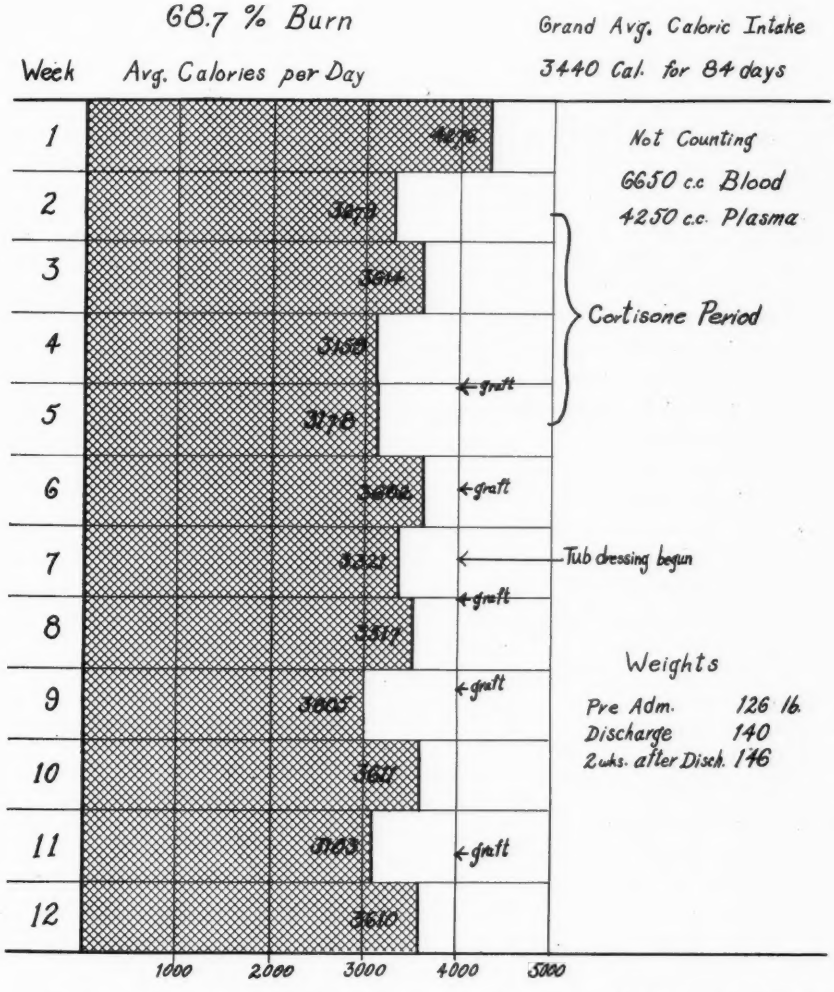

Fig. 5. An extensively burned patient can be maintained on high caloric intake by fluid infusions of 1000 calories per liter solution.

ratio. Other factors were maintained very near equilibrium and the patient was discharged from the hospital the eighth postoperative day.

In burn cases, an accurate electrolyte and fluid balance is impractical. Therefore, the blood chemistries must be observed even more closely. Figure 5 and Table VI represent determinations on a fourteen-year-old girl with a 68 per cent body surface burn, 30 per cent of her body surfaces

High caloric intake was attained by parenteral supplement (Fig. 5), particularly during the first week, of a solution containing 1000 calories per liter (6 per cent amino acids, 12 per cent invert sugar and 5 per cent alcohol). Cortisone therapy was tried for a three-week period as a further attempt to increase her appetite, but no change was found so it was discontinued. We attribute her survival and rapid healing to her youth, balanced

TABLE VI. BLOOD PROFILE—P.B. 68.7 PER CENT BODY SURFACE BURN

Days Post Burn	Adm.	1	1	2	3	4	5	6	7	8	9	10	11	14	16	20	27	34	38	48	58	63	72	76
Hgb.	16.7	19.8	21.8	20.4	15.5	15	16.1	15.5	16.1	17	16.7	16.7	15.8			16.7	14.7	13.3	13.8	15.5	12.6	10.7	11.7	16.7
Hemat.	47.5	58	65	58	46		42	46	46	49	50	42	47			47		39	43					
Rbc.	5, 17		6, 74																					
CO_2		17			25.5	26.4	29.1	26.4	26.4	24.5			27.7			27.8		29.0	30.0					
Cl.		118		104	99.8	99.1	87.5	99.1	99.1	99.1			99.1			99.1		104	95.7					
Na.		147		137	134			136	136					142	143.6	133								
K		4.3		5.4	5.2			4.2	4.5	4.7					5.0	5.9		4.1	4.5					
BUN		8			12			11	12	14			15			15		9	13					
Sugar		332						158		130			117											
Alb.					2.3			2.4		2.5			2.2			2.8		2.3	2.8					
Glob.					2.3			2.5		4.0			4.2			5.0		4.4	3.8					
Total Pro.					4.6			5.9		6.5			6.4			7.8		6.7	6.6					

Normal Range

CO_2	23-29 mEq/L	Prot. A	3.5-5.6 Gm.%
Cl	100-106 mEq/L	G	1.5-2.5
Na	138-146 mEq/L	T	6.8-8.5
K	3.8-5.2 mEq/L	Sugar	80-120 mg.%
BUN	under 20	Hemat.	40-48%

high caloric parenteral and oral feedings, and to careful attention to blood, fluid and electrolyte administration.

The blood chemistry profile is an invaluable guide to therapy (Table VI). Attention may be called to the rise of hemoglobin and hematocrit at the end of the first twenty-four hours. The blood sugar of 332 on the day after admission is no doubt a reflection of the carbohydrate disturbance secondary to stress. We now believe that insulin should be given at that time as an aid in prevention of electrolyte anomalies. The reversal of the A/G ratio persisted, in spite of high caloric intake, until after skin grafting had been completed. During this period repeated whole blood transfusions were needed to correct anemia.

General Rules and Miscellaneous Suggestions

1. Fluid volume management is best accomplished by accurate daily weights which are checked against output measurements and insensible water loss calculation.

2. Fluid and electrolyte balances should be maintained at equilibrium except in correction of a prior deficit (repletion) or when a previous excess is being depressed (depletion).

3. An average preoperative sodium requirement is 105 mEq (6.2 gm.) and 74 mEq (4.4 gm.) in the postoperative period.

4. Potassium requirement averages 45 mEq (3.4 gm.) preoperatively as compared to 67 mEq (5.0 gm.) postoperative.

5. The electrolyte output in pooled samples of urine, suction, etc., is a valuable tool in balanced management. Output values are particularly useful when correlated with serum levels and state of hydration since deficits are recognized prior to drop of serum levels and onset of symptoms.

6. Water should be restricted on the day of surgery to about 2500 cc. above suction losses since the renal response to trauma is one of diminished water output.

7. Blood sugar levels, in the non-diabetic surgical patient, are very valuable since hyperglycemia is commonly associated with electrolyte disturbances. Insulin therapy aids recovery. The so-called pseudo diabetes of trauma is a very common observation in surgical patients. One unit of regular insulin to each four grams of carbohydrate will substantially aid carbohydrate metabolism. Much larger doses are occasionally required.

8. In hypochloremia associated with hypoproteinemia and edema, salt must not be given since the total body chloride quantity is likely normal. The serum chlorides will rise as protein replacement dispels the edema.

9. Hypochloremic alkalosis usually is pathognomonic of potassium deficiency and sodium chloride administration only increases the alkalosis. Serum sodium and potassium are essential laboratory information.

10. In gastric alkalosis due to loss of hydrochloric acid, there has been a great loss of chloride ion without a proportionate loss of sodium. In such cases give 0.6 per cent to 2.0 per cent ammonium chloride, some chloride as potassium chloride, or 6 cc. of concentrated hydrochloric acid (60 — 72 mEq) in 1000 cc. of 5 per cent amino acid solution as a buffer.

11. Ammonium chloride should not be given in the presence of shock because the ammonia ion is very toxic. If the shock state liver does not rapidly convert ammonia to urea toxicity will rapidly appear.

12. Acidosis, with a normal or high chloride, is best managed with 1/6 molar lactate. Again a fairly normal liver function should exist to insure handling of the lactate radical.

13. Electrolyte replacement is best done by adding the desired mEq of each electrolyte from stock concentrates to each liter of basic fluid, thus avoiding the need to order a liter of this and a liter of that.

14. Finally, a high caloric, adequate protein intravenous fluid diet with the addition of the desired electrolytes affords a more smooth and balanced management of the patient's physiologic needs.

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WANTED—A NEW LOOK AT MEDICAL MANPOWER PROBLEMS

Congressional re-evaluation of the physician draft, with more attention paid to the needs of medical education and research, is advocated by Dr. Dean F. Smiley, secretary of the Association of American Medical Colleges, in an editorial published in the March issue of the *Journal of Medical Education*.

Dr. Smiley warns that the nation's potential supply of medical teachers and researchers will be seriously depleted if the present policy of drafting young physicians is continued in its present form. Since the beginning of World War II, Selective Service has delivered as many doctors to the Armed Forces as they estimated they would need.

The vagaries of Selective Service requirements, he says, in some essential respects have been neither fixed nor fair. Interpretations of rulings which vary from state to state, changes in physical qualifications and other factors have tended to keep young physicians, educators and researchers in confusion.

Military service or the threat of it has made long-term planning almost impossible for these men. In addition, more than 3,000 graduate students in the sciences have had their careers interrupted or terminated by military service. "How long," asks Dr. Smiley, "can this nation continue to ignore the need for medical teachers and researchers before we find ourselves falling behind Russia in our ability both to train new physicians and to carry out important life-saving research in such fields as aerophysiology, antibiotic therapy and radiation therapy?"

Complete Obstruction of the Bowel in the Newborn

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BECAUSE many disturbances in the various systems of the newborn result in symptoms that are common to intestinal obstruction, it is often difficult to recognize specific types of obstruction. Yet if obstructions are not recognized early, the newborn may die. Even if the newborn lives long enough for the diagnosis to be made and corrective procedures to be carried out, every hour, even every minute increases the hazard to the newborn.

By limiting these obstructions to the ones obvious shortly after birth, the congenital anomalies become the most frequent source of obstruction. The incidence of various types of intestinal obstruction seen without any limit to age shows that the congenital anomalies are relatively unimportant (Fig. 1).

At St. Mary's Hospital in Minneapolis from 1942 through 1951 there were 24,594 live births. In this group there was a malrotation of the colon, an ileal stenosis with angulation of the bowel, an ileal atresia, a persistent cloaca with a persistent common membrane, and a meconium ileus. That is five congenital anomalies with complete bowel obstruction in 24,594 cases or close to one per 5,000 live births.

1. Congenital atresia of the intestine and colon occurs once in 20,000 births. Although the exact causes of atresia cannot be given, one can safely say they are developmental abnormalities accompanying faulty embryologic events. Ladd and Gross say that such events occur between the fifth and tenth week of fetal life, because before that time and after that time there is a rapid proliferation of epithelium from the pylorus to the ileocecal valve which obliterates the lumen. The formation of vacuoles and then the coalescence of these cystic spaces re-establishes the lumen. Arrest of growth at this period anywhere from the pyloric sphincter to the ileocecal valve may ensue in an atresia. The most frequent site of such an obstruction is in the lower ileum and jejunum. In 392 cases collected by Davis and Poynter, 161 were in the jejunum and ileum.

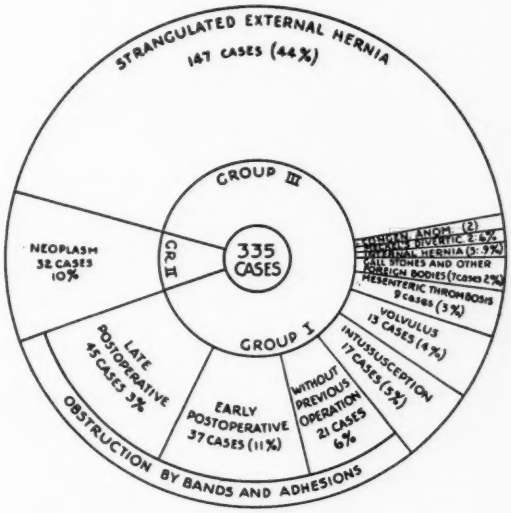

Fig. 1. The incidence of various types of intestinal obstruction, over a ten-year period at the Massachusetts General Hospital, during which time there were 39,936 surgical admissions, 335 cases of bowel obstruction were seen. The high incidence of strangulated hernia in this series is striking. (Reprinted by permission of Wangensteen, O. H., from "Intestinal Obstruction", ed. 2).[7]

Figure 2, taken from Ladd and Gross, shows the usual distribution of sites of obstruction. The obstruction is usually single but occurs in multiple areas in 15 per cent of the cases.

The obstruction may occur in two forms. There may be an internal diaphragm or veil which completely blocks the lumen, or there may be a blind sac of proximal intestine connected to the caudal section by a fibrous band. Above the obstruction, the bowel may be so distended as to rupture. Below the obstruction the bowel is collapsed and ribbon-like, especially in low obstructions.

When the obstruction occurs high in the intestinal tract the child usually has symptoms

Inaugural Thesis presented before the Minneapolis Academy of Medicine, November 17, 1953, Minneapolis, Minnesota.

early. They always occur within the first day of life. Vomiting which becomes more and more frequent and intense is a major symptom. There may or may not be bile in the vomitus, depending

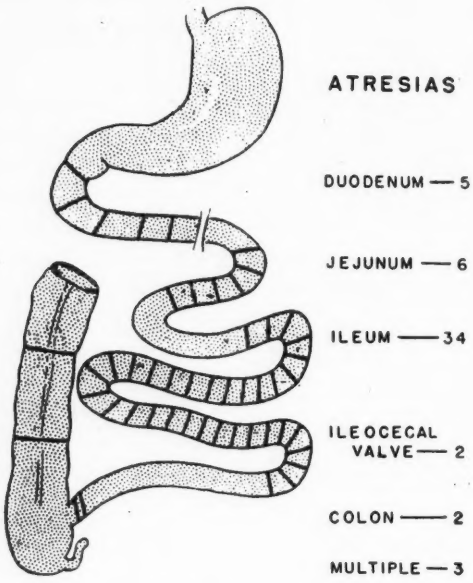

Fig. 2. Diagram showing position of atresias in fifty-two cases. (Reprinted by permission of Gross, Robert, from "Abdominal Surgery of Infancy and Childhood").[2]

on the level of the obstruction. Obstructions above the papilla of Vater are rare, so that nearly all such infants have bile-stained emesis. The stools are usually scanty in amount and drier than normal. The color is usually not so tarry. The degree of abdominal distention depends upon the level of the atresia. A high obstruction may show little distention because the baby can keep the stomach empty by vomiting. An atresia in the jejunum will show marked and general distention due to gas in the bowel above the obstruction which is not expelled with vomiting. A low atresia often shows intestinal patterning. If fever is present, it is usually due to dehydration and/or peritonitis following rupture of the bowel.

Because of its aid in locating the point of obstruction, roentgen examination is important. Gas in the stomach and duodenum, without gas in the lower intestine, locates the obstruction in the duodenum. A thin mixture of barium or lipiodol orally will locate a high obstruction, and a barium enema will locate an obstruction in the colon. Actually, the use of contrast media is seldom needed, and to avoid the danger of aspiration, lipiodal is preferred.

Farber's test will help one determine if the obstruction is complete or partial, and also if it has been present since before birth. The meconium is examined and the presence of vernix cells (cornified epithelial cells) can quickly be established.

A low bowel obstruction can be differentiated from imperforate anus by external inspection and the passing of a bougie or a barium enema. A high bowel atresia can be differentiated from internal herniation of the bowel or volvulus by absence of bile in the meconium. Also, the abdomen is flaccid and non-tender in an atresia, in contrast to tenderness in internal herniation or volvulus.

2. Malrotation of the intestine and colon is much more complicated than atresia as to etiology. To try to visualize it quickly and yet clearly, picture the gut first as a long tube and then as a tube with a "U" shaped piece but still in a single plane suspended by a common dorsal mesentery. Now think what would happen when one section of this tube grows more rapidly than another. It would either have to extend a greater distance, which it can't do because of it's abdominal limitation, or twist to take up the slack. To add to the slack, the "U" shape which is the portion of the intestine protruding through the umbilical orifice now returns to the abdominal cavity. This is at the tenth week of fetal life. The position that various parts of the intestinal tract will take in relation to each other is the result of the intestine rotating in a counter-clockwise manner; and because the cecum is the last part to return to the abdomen, the colon is carried forward over the superior mesenteric artery.

The malrotation may be due to non-rotation of the midgut loop, reversed rotation of the midgut loop, or some other abnormal rotation of the midgut loop. In reversed rotation, the transverse colon lies behind the superior mesenteric artery, giving rise to constricting bands, and there is a lack of attachment of the mesentry along the posterior abdominal wall, predisposing to volvulus.

There are two main features of a malrotated bowel. One is obstruction near the duodeno-je-

junal angle occasioned by abnormal adhesions, that is, bands lying directly across the descending portion of the duodenum and obstructing by extrinsic pressure. The other is a volvulus of the small bowel, cecum, and ascending colon or torsion about the superior mesenteric artery. The symptoms are vomiting, abdominal distention, peristolic waves, dehydration, and fever. X-ray will show a gas-filled stomach and duodenum. If there are no bands across the duodenum, there will be an enormously dilated jejunum and ileum. A barium enema will show the cecum in the right epigastrium in an incompletely rotated position. Laboratory findings are of little or no importance. The only change is a change consistent with dehydration. It may be difficult at times to differentiate this from intestinal atresia, but once it has been established that surgery is necessary, this point is not too important.

3. An imperforate anus occurs once in 5,000 newly born babies. In the embryo, there is a period when the urogenital and intestinal tracts open into a common cavity, the cloaca. This is closed from the exterior by a cloacal membrane. As the embryo develops there is a cleavage of the urogenital tract from the intestine. The two are entirely separate at the seventh week (Fig. 3). Late in the seventh week the urogenital membrane, the anterior part of the original cloacal membrane, opens; and later the anal membrane (the posterior part) opens. Arrest in development at this stage results in an imperforate anus or a stenosis. There are four types of anal anomalies (Fig. 4). These are:

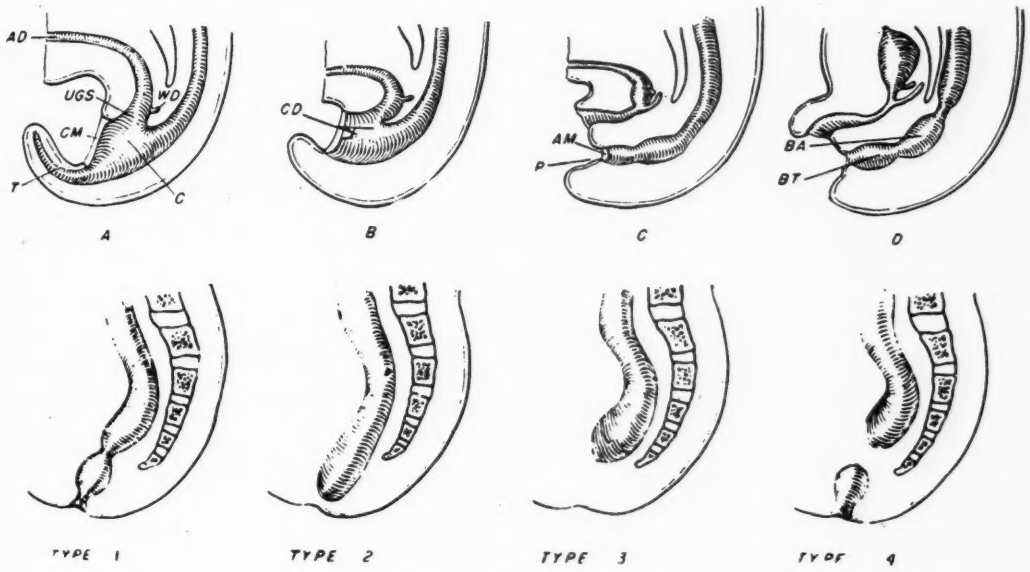

Fig. 3. *(Above)* Normal development of the anus and rectum. A, 7.5 mm. stage. B, 9 mm. stage. C, 22 mm. stage. D, 42 mm. stage.

AD, Allantoic Duct	C, Cloaca	P, Proctodeum
AM, Anal Membrane	CD, Cloacal Duct	T, Tail Gut
BA, Bulbus Analis	CM, Cloacal Membrane	UGS, Urogenital Sinus
BT, Bulbus Terminalis		WD, Wolffian Duct

(Reprinted by permission of Gross, Robert, from "Abdominal Surgery of Infancy and Childhood".)[2]

Fig. 4. *(Below)* Types of anal and rectal abnormalities. Type 1—Stenosis at anus or at a point several centimeters above the anus. Type 2—Imperforate anus. Obstruction due to persistent membrane. Type 3.—Imperforate anus. Rectal pouch ending blindly some distance above anus. Type 4—Anus and anal pouch normal. Rectal pouch ends blindly in hollow of sacrum. (Reprinted by permission of Gross, Robert, from "Abdominal Surgery of Infancy and Childhood.")[2]

1. Stenosis of anus or just above anus,
2. Imperforate anus,
3. Imperforate anus with rectal pouch ending blindly, and
4. Anus normal with rectal pouch ending blindly.

4. Extrinsic bands, aside from those associated with malrotation of the gut and post-surgical complications, are rare. However, one special type will be discussed, the annular pancreas. This obstruction is due to a complete encirclement of the duodenum by a ring of pancreatic tissue. The

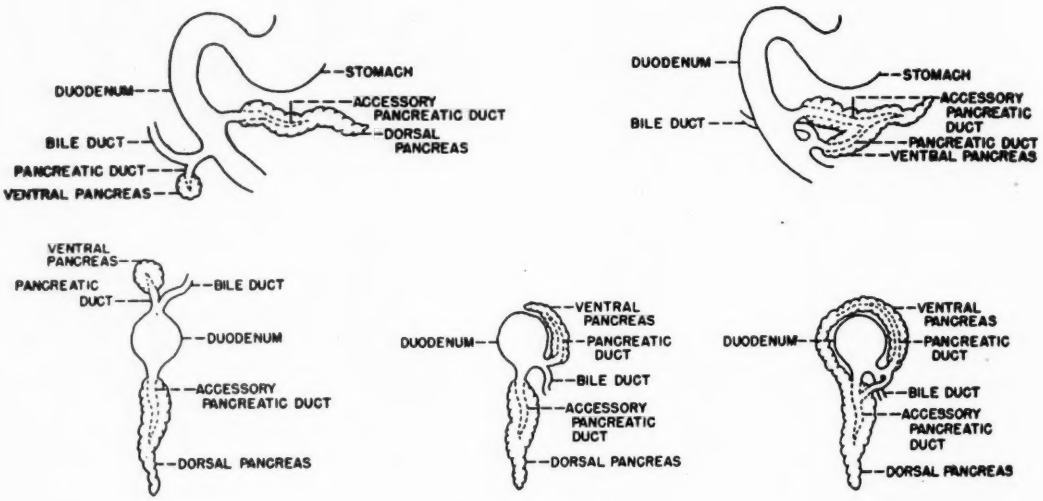

Fig. 5. *(Above) (left)* Pancreas of human embryo of five weeks (Kollman). *(Right)* Pancreas of human embryo at end of sixth week (Kollman). Courtesy, Gray's Anatomy. (Reprinted by permission of May, Charles D., editor of *Pediatrics*)[4]

Fig. 6. *(Below)* Diagrammatic representation of mode of development of annular pancreas. Free end of ventral pancreatic anlage becomes fixed so that during its subsequent migration, with rotation of duodenum toward dorsal anlage it is drawn out. With fusion as shown at right, it comes to surround duodenum (after Lecco). (Reprinted by permission of May, Charles D., editor of *Pediatrics*)[5]

The most difficult to diagnose is type four. The easiest types to treat are types one and two.

No type of bowel obstruction is more easily recognized than the first three types. Clinically a child will take feedings for at least twenty-four hours before regurgitation begins. The external recognition of the imperforate anus is apt to be discovered before any obstructive symptoms develop. The x-ray is a big help in determining the type of obstruction present. The Wangensteen-Rice maneuver is a help in locating the height of the blind pouch. An x-ray is taken of the infant in an upsidedown position. The height the gas ascends will determine the level of the blind pouch. One must not rely on this method before twenty-four hours have elapsed to allow time for the gas to pass through the sticky meconium. About twenty-eight per cent of these children have associated anomalies.

majority of individuals with an annular pancreas do not develop symptoms until they have reached adult life, or they may never develop symptoms and the finding is one at the autopsy.

Normally, the pancreas develops as two distinct entodermal diverticuli known as the dorsal and ventral anlage. The dorsal anlage becomes the body and tail of the pancreas. The ventral anlage comes off the primitive bile duct and forms the head of the pancreas. At six weeks in fetal life the two portions fuse. This is the period when the anomaly arises (Fig. 5). To cause obstruction, one of three situations arises. First, if the ventral anlage does not rotate to its normal posterior position there will be pancreatic tissue on both sides of the duodenum. Second, if the ventral anlage becomes adherent to the duodenum, the rotating of the duodenum merely stretches the ventral anlage to form a ring. Third, an unusual

overgrowth of one of the ducts may lead to complete encirclement of the duodenum (Fig. 6).

In this condition there is vomiting, upper abdominal distention and visible persistaltic waves, decreased size and frequency of meconium stools, progressive weight loss, and dehydration. On x-ray there is a dilated stomach and dilated proximal duodenum. Barium studies are not necessary.

5. Another congenital anomaly is *agenesis of the myenteric plexus*. These children have vomiting, constipation and abdominal distention during the first four days of life. The picture is more like a lower bowel obstruction. The x-ray will show fluid levels if taken in the upright position. A barium enema will help locate the gas and will show *normal* and dilated bowel. Although the junction between normal and dilated bowel is usually the point of change from abnormal to normal innervation, one cannot accept the roentgenographic findings or the operative findings as evidence of this junction. There may be a considerable segment of dilated bowel that contains no ganglia. The normal or spastic appearing bowel is the area of abnormality (Fig. 7).

6. In an infant with *meconium ileus* the entire intestine is full of tough and inspissated meconium. The end result is obstruction and fairly often a perforation of the intestinal wall. On autopsy, obstruction of the bile duct, the pancreatic duct or both is a finding. Any child that has this probably has cystic fibrosis of the pancreas. The symptoms are the same as in high obstruction—vomiting, abdominal distention and absence of stools.

Case 1.—B.G.H. was a girl born August 21, 1950. She weighed 2.67 kilograms and had a normal delivery. Her postnatal examination on August 22, 1950, was essentially normal, no vomiting or abdominal distention. Vomiting started August 23, two days after delivery. Atropine sulfate was ordered to be given before feedings and subcutaneous fluids were ordered. The child had a meconium stool on August 23, 24 and 25. No more stools were recorded to the date of surgery, August 31.

Fluoroscopic examination on August 28 (seven days after birth) showed a barium swallow progress to the mid-duodenum. The impression was partial obstruction on the basis of a volvulus. Some of the barium passed through. A barium study of the colon showed incomplete rotation of the colon with the major portion of the colon in the left side of the abdomen. An operation, done August 31, revealed the cecum and ascending colon in the left upper quadrant, wrapped around the root of a midgut volvulus in a 360 degree turn.

There were transduodenal bands extrinsic in location across the third and fourth portions of the duodenum. There were a few additional bands over the proximal jejumen. After untwisting the bowels and cutting the bands, the wound was closed. The child is now two years and two months old and doing well.

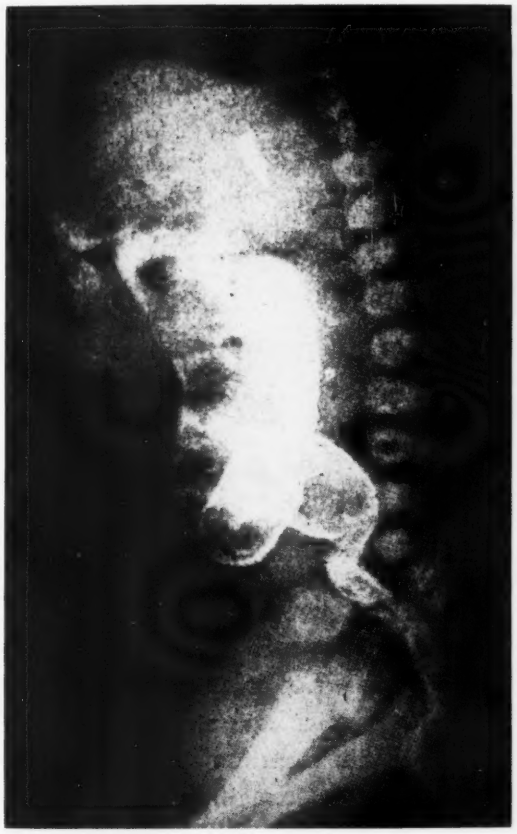

Fig. 7. Small barium enema illustrates narrow spastic rectum and rectosigmoid with dilated bowel above. (Reprinted by permission of May, Charles D., editor of *Pediatrics.*)[5]

Case 2—B.G.T. was a girl born October 15, 1950. The child was cyanotic at birth but pinked up rapidly with oxygen. She weighed 2.14 kilograms. One thing that was misleading to the doctor who saw the case originally is that the child did not vomit apparently large amounts, although the weight loss was 373 grams in four days. The explanation of the peculiar type of vomiting was discovered when the baby was lavaged and 300 cc. of fluid was removed from the stomach. On October 20, 1950, the baby was dehydrated, jaundiced, lethargic and had a distended abdomen. The x-ray showed a marked distention of the stomach with fluid and gas compatible with a high grade obstruction in the region of the pylorus. An operation done the same day, October 20,

(Continued on Page 182)

ACH

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Blood Volume Studies In Gastrointestinal Hemorrhage

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Minneapolis, Minnesota

EVEN THOUGH standardization of diagnostic procedures and therapy is a goal sought after by most of us in the medical profession, it should not be assumed by any of us that similar lesions should be handled in the same manner by all surgeons anymore than we should assume that all surgeons have equal facilities and skill. This fallacy seems to be especially prevalent in the discussions of the management of gastrointestinal bleeding. The procedure of choice in each case, whether in diagnosis or treatment, is that which yields the best results in that surgeon's hands. An attempt will be made here to bring out a point which should better assist one in evaluating the condition of a gastrointestinal bleeder.

We have all seen patients with gastrointestinal bleeding who we know have bled as much as one-third or one-half of their total blood volume and still do not exhibit the classical signs of shock as we know it. On the other hand, why are some patients with considerably less bleeding in profound shock when we see them. The answer, in most cases, appears to be in the total blood volume and red cell mass studies before the bleeding starts and after we see that patient.

Table I shows the normal values for blood volume, red cell mass and plasma volume in a 70 Kg. male with a hematocrit of 46 per cent and a hemoglobin of 16 gm. If this man should suddenly lose one-third of his blood volume by hemorrhage, he would then have remaining the values shown in Table II. This total of 4000 cc. blood volume is only theoretical because we know that along with the vascular constriction that takes place, there is also an exchange of fluid from the interstitial spaces into the vascular bed so that we would have a volume of, say, 5000 cc. The number of red cells is decreased by one-third so that the hemoglobin and hematocrit are lowered.

Table III shows how this affects the plasma volume, hemoglobin and hematocrit. This hemoglobin of 12.8 gm. and hematocrit of 37.3 per cent are not far off the normal and may not cause us much alarm, especially if we think the bleeding has stopped. However, most of the patients with gastrointestinal hemorrhage are still bleeding when we see them and may continue to bleed for some time during our care. Also we know that a good percentage of these patients will bleed again, and each time they bleed there is a further loss of total hemoglobin from the circulating blood.

In a similar manner the circulating proteins will be reduced from a normal figure of 6.0 gm. per 100 cc. blood to a corresponding low after the volume is reduced and then diluted as it increases. Table IV shows the effect on total plasma proteins.

From these figures on a hypothetical case, it can be readily seen that further hemorrhage at this

TABLE I. NORMAL VALUES FOR A 70 KG. MALE WITH A HEMOGLOBIN OF 16 GM AND A HEMATOCRIT OF 46 PER CENT.

Blood Volume	Weight in Kg. x 85	6000 *cc*
Plasma Volume	Weight in Kg. x 45	3200 *cc*
Red Cell Mass	Weight in Kg. x 40	2800 *cc*
Total Hemoglobin	$16\text{x}\ \frac{6000}{100}$	960 Gms.

46% of 6000—2800 *cc* red cell mass
6000—2800 *cc*——3200 *cc*——plasma volume

TABLE II. VALUES FOR A 70 KG. MALE WHO HAS HAD A SUDDEN LOSS OF ONE-THIRD OF HIS BLOOD VOLUME BY HEMORRHAGE.

Blood Volume	4000 *cc*
Plasma Volume	2133 *cc*
Red Cell Volume	1867 *cc*

Hemoglobin—16 Gms/100 *cc*—640 Gms total.
Hematocrit—46%.

TABLE III. VALUES AFTER EXCHANGE OF FLUID BETWEEN VASCULAR BED AND INTERSTITIAL SPACES.

Blood Volume	5000 *cc*
Plasma Volume	3133 *cc*
Red Cell Volume	1867 *cc*
Total Hemoglobin	640 Gms.

Hemoglobin per 100 *cc*=12.8 Gms.
Hematocrit—37.3%.

Read before the Minneapolis Academy of Medicine, March 16, 1954.

TABLE IV. PROTEIN VALUES BEFORE AND AFTER THE HEMORRHAGE.

Total Proteins .....6.0 Gms./100 *cc*
or Plasma Volume = 3200 x 6 = 198 Gms. total protein.

100 100

After the bleeding
2133 x 6 = 128 Gms. total protein.

100

After increase of plasma volume to 3133 *cc* the total protein is still 128 Gms. or 4.1 Gms./100 *cc*.

time or even after inadequate transfusions of whole blood will seriously jeopardize this patient's life. This is especially true if surgery is contemplated on this patient because with the added trauma of surgery, anesthesia and surgical hemorrhage, the seriousness of this situation could become alarming.

Of equal importance in the therapy of hemorrhage is the condition of that patient with respect to blood volume and red cell mass before he starts bleeding. These patients may have depleted values in blood volume as a result of nutritional disturbances, heart disease, chronic infection, slow chronic bleeding or malignancy. For purposes of simplification it might be said that this type of patient is suffering from "chronic shock."

This chronic shock has been defined as a syndrome characterized by weight loss, diminished blood volume, increased interstitial fluid volume and an increased susceptibility to shock from anesthesia, surgical trauma, or a physiological shock condition such as hemorrhage.

It is readily apparent from the above example of change in blood volume, red cell mass and plasma proteins that profuse bleeding is much more likely to cause the patient to fall into a shock-like state than a normal healthy individual.

The total protein will be especially affected because during this subpar state the patient is continually using his supply of stored protein and in any emergency he would not have this supply to fall back upon in order to maintain an adequate plasma protein value.

From this, it is quite obvious that the patient with rapid bleeding will show close to normal hemoglobin, hematocrit and plasma protein values. However, in the patient with the same amount of bleeding over a longer period of time, these values are lower than normal. In this type of patient, the plasma volume will be near normal and on receiving transfusions, this plasma volume changes very little. The mechanism of this phenomenon is that there is an exchange of fluid between the circulating blood and the extracellular spaces. In this case the goal should be replacement of the red cell mass and not the blood volume.

The test most used at this time for the determination of the plasma volume is the Evans blue dye method. This involves the intravenous injection of the dye. After ten minutes a specimen of blood is obtained from the opposite side of the body. No tourniquet is used. The plasma from the centrifuged blood is then compared by a colorimetric method with a photo-electric spectrophotometer. This method has been shown to be accurate to within 3 per cent. Another test which has been used more recently is the use of tagged radioactive ions. This is thought to be more accurate than the Evans blue dye method but for all practical purposes the dye method is probably accurate enough.

In conclusion, I would like to leave the following facts to be noted whenever one is confronted with a patient on whom surgery is contemplated and who has experienced hemorrhage either slow or fast or, for other reasons mentioned, presumably the plasma volume or red cell mass is altered.

1. The hemoglobin and hematocrit levels and other clinical signs or lack of signs of shock are not accurate guides to severity of hemorrhage.

2. The red cell mass is a more important determination to know than the hemoglobin and hematocrit in order that a true evaluation of the patient's condition may be known.

3. Because there are shifts in fluids between the plasma and the interstitial space and also at the same time shifts between the interstitial space and the intercellular space, it would be better to defer surgery in many cases until adjustment has been reached.

4. Because the shift of these fluids is not a rapid process but is not complete for at least twenty-four hours, it is probably better to defer surgery until the patient has had time to adapt to his increased blood volume and to his electrolyte fluid balance. Also because of this, transfusion should not be pushed too rapidly unless the patient is in clinical shock. There is always the danger of overloading the circulation and throwing the patient into cardiac failure with pulmonary edema and the subsequent complications.

408 Physicians and Surgeons Building

Gynecological Cancer Detection

Findings at University of Minnesota Cancer Detection Center

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and
MAXWELL M. BARR, M.D.
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IN THE PAST two decades increasing emphasis has been placed on the early detection of carcinoma. The importance of periodic examinations and the recognition of early symptoms have been the basis of educational programs directed at the medical profession as well as the lay public. As a by-product of these efforts, over 133 cancer detection centers have been established in this country since 1937.[1,8] The Cancer Detection Center at the University of Minnesota was established in March, 1948, with the approval of the Minnesota State Medical Association. Examinations have been limited to men and women over the age of forty-five years. The scope of the examinations and the results obtained have been reported elsewhere.[5,7,10]

This report deals with the results of gynecological examinations and special diagnostic procedures performed upon 3,087 women from March, 1948 to October 6, 1953. During this period the following routine has been used for the systematic examination of the female pelvis: (1) A brief history is obtained to elicit the presence of abnormal bleeding or discharge. (2) The lower abdomen is inspected and palpated. (3) The external genitalia are inspected and palpated. (4) Vaginal secretion is aspirated for the vaginal smear. (5) Speculum examination of the vagina and cervix is performed and a cervical smear is taken with an Ayres' wooden spatula. Since 1950 an additional routine has included the insertion of a small piece of gelfoam into the endocervix. If bleeding results the gelfoam is placed in formalin and sent to the pathology laboratory for preparation of sections similar to tissue biopsy. The slide is examined by the pathologist for the presence of abnormal cells in the interstices of the gelfoam as described by Gladstone.[3] (6) Bimanual examination and recto-vaginal examinations are performed.

In addition to the above routine, the following procedures are performed when indicated: (1) Punch biopsy of the cervix is taken when an abnormal lesion or bleeding area is found. When the Papanicolaou studies are reported positive or suspicious, repeated smears are made. If these are positive, biopsies are taken from target areas indicated by Schiller's Gram iodine test. If target areas are not found, biopsies are taken in the four quadrants of the cervix, or a cone of cervical tissue is removed with a Gusberg cone biopsy instrument. (2)) Small cervical polyps are avulsed with Bozeman uterine dressing forceps or the Pratt forceps. When a history of abnormal bleeding is obtained, the patient is referred back to her own physician for dilatation and curettage. (3) When cervical smears are positive but biopsies of the cervix fail to reveal carcinoma, the patient is referred to her physician for dilatation and curettage and either repeat biopsies or knife conization of the cervix. (4) When other lesions or pelvic masses are found, the patient is referred to her physician for further observation or therapy as indicated. (5) Endometrial aspiration smears have more recently been made on patients with abnormal uterine bleeding or discharge in an attempt to improve the screening of patients with endometrial carcinoma. Figure 1 illustrates some of the instruments used for routine and special procedures during the gynecological examination.

The vaginal and cervical smears are stained and

Presented before the Minneapolis Academy of Medicine, April 20, 1953.

This material includes examinations through October 6, 1953, or five and one-half years of operation of the center. The authors wish to acknowledge with thanks the co-operation of Dr. Claude R. Hitchcock, director of the center, in making the records and facilities available for this study. Mrs. Lydia Linsey has been most helpful in assembling data necessary for this report.

The authors have attended the center as gynecological examiners since its inception. The Cancer Detection Center is sponsored by a grant from the Minnesota Division of the American Cancer Society.

TABLE I

	Number	Per cent
Female patients (all over age 45)	3,087	
Total examinations	6,840	
Cervical biopsies	419	13.5
Gelfoam saved	563	
Pap. smears	10,334	
Cervical polyps	193	6.2
Carcinomas found in polyps	2	
Carcinomas found in cervical biopsies	6	
Total—carcinoma of the cervix	8	0.26

TABLE II

	Number	Per cent
Total Pap. smears	10,334	
Total patients	3,087	
Total examinations	6,840	
Class 3 smears	109 smears in 72 patients	
Class 4 smears	14 smears in 10 patients	
False pos. class 3	68 patients	
False pos. class 4	3 patients	
Total false pos.—classes 3 and 4	71 patients	2.3
Total false negative	3 patients	0.1

TABLE III

	Number
Normal	2
Cystic nodule	1
Friable polyp	1
Roughened post. lip	1
Bleeding spot	1
Polypoid tissue	1
Localized granular erosion	1

examined by the technicians in the laboratory of the Cancer Detection Center. Suspicious smears are examined by the pathologist in the University Hospitals for final classification. Technician screening has resulted in a considerable saving of valuable time on the part of professional personnel.

Cancer of the Cervix

Table I shows the number of special examinations performed in the group of 3,087 patients examined from March 1, 1948, to October 6, 1953. The average number of examinations per patient was 2.2 during this five and one-half year period. One hundred and ninety-three cervical polyps were removed, and malignant degeneration was present in two.

Punch biopsies of the cervix were positive for carcinoma in six women. A total of eight carcinomas of the cervix have been detected among women who have attended the center (.26 per cent).

The experience with Papanicolaou smears is shown in Table II. The smears have been classified as follows: Class 1, normal appearing epithelium; Class 2, excessive number of pus cells; Class 3, atypical epithelial cells which are not frankly anaplastic in appearance; Class 4, frankly anaplastic appearing cells.

The relatively high percentage of false positive smears should decrease with increasing experience in reading the smears. Graham and Meigs[4] have shown that their group was able to reduce the percentage of false positive smears from 6.4 per cent to .04 per cent in a period of five years.

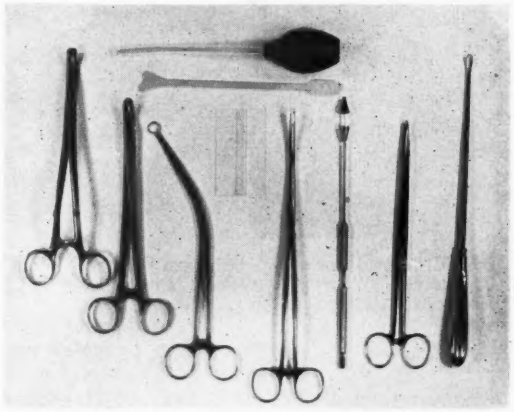

Fig. 1. Instruments used for detection of carcinoma of the internal female genitalia. Left to right are Thoms modification of the Gaylor biopsy punch, Gaylor biopsy punch, single tooth tenaculum, uterine dressing forceps holding gelfoam, Gusberg cone biopsy, Pratt forceps, and a small sharp curette. Above are a glass canula for aspirating vaginal secretion and the Ayres wooden cervical spatula.

The appearance of the cervix in the group of eight women with carcinoma of the cervix is described in Table III.

These findings indicate the lack of any characteristic gross appearance that will definitely identify early carcinoma of the cervix. Two patients with normal appearance of the cervix were detected with Papanicolaou smears graded Class 4. The smear diagnosis was confirmed by biopsy in each case.

Case 1.—M.C.O., aged sixty-two, para 3. At her third visit to the center her vaginal smear was found to be Class 4. Previous smears were Class 1. The cervix appeared normal. The vaginal smear (Fig. 2) revealed a typical anaplastic appearance. A biopsy obtained by her physician revealed early squamous cell carcinoma with no evidence of invasion.

Case 2.—P.F., aged fifty-four, para 2. At her first visit to the center her cervix appeared normal but the vaginal smear was Class 3. Repeat smears over a three-month period revealed Class 3 and later Class 4 smears. Schiller's test was positive around the circumference of

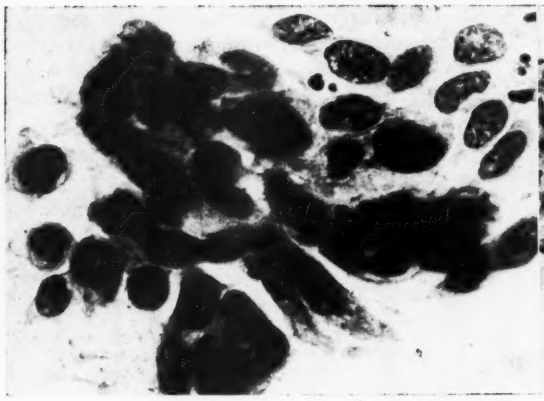

Fig. 2. Vaginal smear obtained from Case 1. Normal appearing cervix. Smear is Class 4. Biopsy taken elsewhere revealed squamous cell carcinoma.

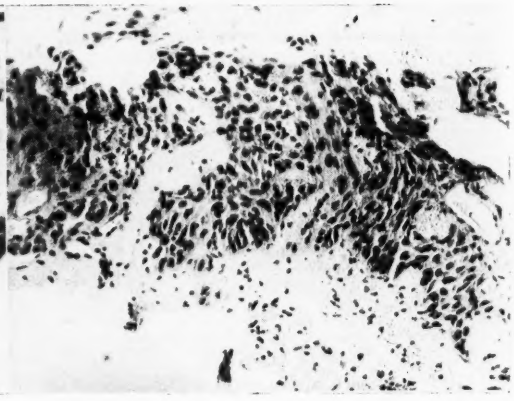

Fig. 3. Biopsy of cervix from Case 2. Normal appearing cervix. Positive Schiller's test after Class 4 smears. Gusberg cone biopsy reveals invasive squamous cell carcinoma.

the vaginal cervical junction. A biopsy was obtained with the Gusberg cone punch and was reported as showing invasive carcinoma of the cervix (Fig. 3).

The remaining six cases of carcinoma of the cervix were diagnosed by biopsy of abnormal lesions of the cervix. However, the routine Papanicolaou studies revealed Class 4 smears in three patients, a Class 3 smear in one patient, and normal smears in one patient. Figure 4 shows a Class 4 cervical smear obtained from a fifty-three-year-old woman, para 2. Biopsy of a small localized granular erosion of the cervix revealed early squamous carcinoma (Fig. 5). In six of the eight patients with positive cervical biopsies no evidence of invasion was found in the tissue examined. The following case is of interest because it was detected only by a set of fortunate circumstances:

Case 3.—E.A., aged forty-five, para 3. At her second visit to the center a biopsy was taken of a small area of the cervico-vaginal junction that bled easily. The tissue was examined by two pathologists, both of whom diagnosed chronic cervicitis. One report noted the presence of a few suspicious looking cells at the margin of the tissue section. Repeat biopsy was requested and was done two weeks later, several small bites being taken from the site of the previous biopsy. This area was located easily because the first biopsy site had been sketched by the examiner. The diagnosis now was definitely squamous cell carcinoma of the cervix (Fig. 6).

The gelfoam biopsy was saved and examined in five of the eight women found to have carcinoma of the cervix. These were interpreted as positive for carcinoma in one patient (Fig. 7), suspicious in three patients, and negative in one. The gelfoam test was not responsble for detecting carcinoma of the cervix but was found confirmatory in four of the five patients in whom the test was used.

Carcinoma of the Endometrium

Two women were found to have carcinoma of the corpus uteri during the first five and one-half years of the detection center's operation. Both presented the history of postmenopausal bleeding and both were referred to their own physicians for diagnostic curettage, which revealed the lesions. In both patients the pelvic findings were normal to bimanual examination and the surgical specimens revealed well localized lesions. Vaginal and cervical Papanicolaou smears were negative in both cases. This experience is similar to that at other centers and has led to the use of endometrial smears as a screening method.[9] Our experience with the endometrial smear is too recent for evaluation.

Carcinoma of the Ovary

Adnexal masses were palpated in 100 women. Of these, forty-five patients are known to have had pelvic laparotomy. Five patients were found to have carcinoma of the ovary. Four of these women were asymptomatic, and one complained of pelvic pain. Cystadenocarcinoma was found in four women, and one had primary adenocarcinoma. On September 1, 1954, four women were alive at four and one-half, three and one-half, three, and two years after surgery, and one patient had expired. Although cure is the excep-

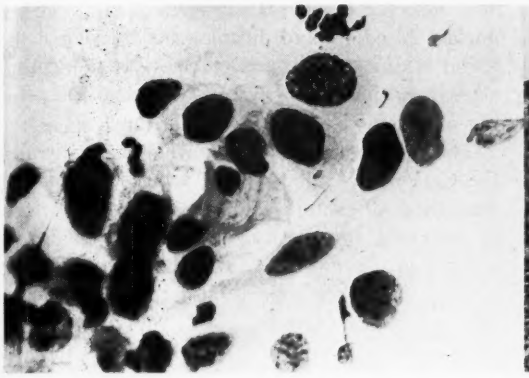

Fig. 4. Cervical smear, Class 4, from cervix with small localized granular erosion.

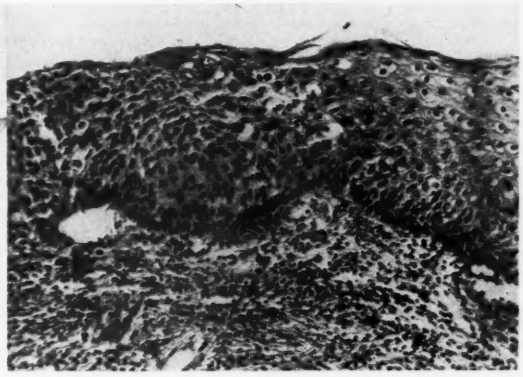

Fig. 5. Biopsy of small granular erosion shows early squamous cell carcinoma. Same woman as in Figure 4.

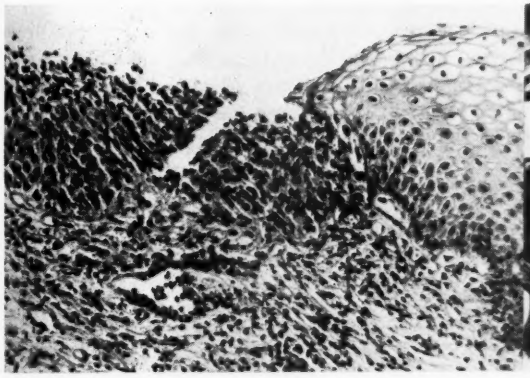

Fig. 6. Biopsy from small bleeding area of cervix repeated at request of pathologist. Squamous cell carcinoma.

Fig. 7. Gelfoam biopsy of cervix from patient with small cystic nodule of cervix found to contain invasive squamous cell carcinoma.

tion, the early detection of carcinoma of the ovary offers an opportunity for adequate surgical therapy and prolongation of life.

Precancerous Lesions of the Female Pelvis

Probably the only disease process in the female pelvis that is associated with carcinoma in a significant percentage of women is leucoplakic vulvitis. This condition, as its name implies, presents a characteristic white thickening of the vulvar skin which is often confused with an exaggerated aging process of the vulvar skin known as kraurosis vulvae or, literally, shrinkage of the vulva. Kraurosis carries no increased threat of carcinoma while leukoplakia must be observed for the development of ulcers, fissures and induration of the involved skin. Biopsy will reveal the onset of carcinoma of the vulva. Patients with leukoplakic vulvitis have been referred to their physicians with the recommendation that they be examined every six months and biopsies taken as indicated. No carcinoma of the vulva has been detected at the center to date.

Myomata of the uterus may become sarcomatous in a small but definite percentage of women. The incidence of sarcomatous degeneration has usually been reported between 0.5 per cent and 1.0 per cent of all women with myomata.[11] In the general population this figure may be as low as 0.2 per cent. Sudden growth of these tumors, especially after the menopause, should indicate the danger of this complication.

Chronic cervicitis, leucoplakia of the cervix, cervical polyps and urethral caruncles are not

TABLE IV

	Number	Per cent
Skin	18	
Breast	7	
Stomach	5	
Sigmoid	3	
Rectum	3	
Cecum	2	
Duodenum	1	
Thyroid	1	
Lung	1	
Leukemia	1	
Lymphosarcoma	1	
Total Extrapelvic	43	1.39
Total Pelvic	15	.48
Total malignancies 3,087 women	58	1.87

precancerous but may mask or be simulated by the appearance of early carcinoma. Figures available for the first four years of operation of the center reveal that 381 women (17.5 per cent) of the female population attending the center have had hysterectomies. The cervical stump was left in 275 women or 12.7 per cent of the total. Neither carcinoma in the cervical stump nor sarcomatous degeneration of a myoma has been found in any detection center patient.

Extrapelvic Cancer

Carcinoma was detected outside of the pelvis in forty-five women during the five and one-half years of this study. The location and number of these malignant tumors or conditions are indicated in Table IV.

Discussion

During the first five and one-half years of operation of the University of Minnesota Cancer Detection Center, 3,087 women over the age of forty-five years received 6,840 examinations. Eight women were found to have carcinoma of the cervix, two had carcinoma of the endometrium, and five had carcinoma of the ovary. Extrapelvic carcinoma was found in 43 women.

Biopsy of abnormal appearing areas of the cervix was responsible for detecting six of the eight carcinomas of the cervix. Cytological smear study was responsible for detecting two carcinomas of the cervix. Although the Papanicolaou studies have proved to be an important means of screening for carcinoma of the cervix, the current shortage of technicians capable of screening smears makes general usage of this method impracticable except in large centers of population. The gelfoam method of cytological study has not been responsible for detecting carcinoma but has indicated the need for further study in four of five cases. Carcinoma of the endometrium has been detected only by eliciting the history of abnormal bleeding and histologically proving the lesion after dilatation and curettage. Endometrial aspiration smears give promise of effective screening of carcinoma of the endometrium.

The follow-up of women who have attended the Cancer Detection Center is accomplished by contacting all examinees once each year in regard to returning for check-up. In this manner close contact is maintained with greater than 90 per cent of the center population. Up to the time of this report no examinee who was found to be normal on gynecological examination has been found on follow-up to have developed pelvic malignancy, with the possible exception of one patient who was subsequently found to have generalized lymphosarcoma of the abdominal viscera at laparotomy.

The accuracy of the screening pelvic examination to date would indicate that careful periodic examination and biopsy of all suspicious lesions will detect carcinoma of the vulva, vagina, cervix and ovaries in a high percentage of affected women. The Cancer Detection Center is valuable for testing and teaching new methods but cannot substitute for the doctor's office in reaching the bulk of the population.

Conclusions

1. Carcinoma of the cervix can be detected early by careful periodic examination and biopsy of all suspicious lesions. Vaginal and cervical smears are an important adjunct for early detection. Gelfoam biopsies have not proved valuable in this study.

2. Carcinoma of the endometrium can be detected early if women are educated to see their physicians for abnormal bleeding and the physicians follow through with adequate differential curettage. Endometrial aspiration smears offer better promise of effectively screening carcinoma of the endomerium than do vaginal and cervical smears.

3. Carcinoma of the ovary can be detected early only by periodic pelvic examinations and exploratory laparotomy for all newly developed and enlarging adnexal masses.

4. Carcinoma of the vulva is rare but will be detected early by biopsy of all suspicious lesions of the vulva particularly when the lesions are associated with leukoplakic vulvitis.

5. Significant reduction in mortality rates will

(Continued on Page 188)

Ectopic Pregnancy

Diagnostic Analysis of Seventy Cases

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DURING the thirteen and one-half years from January, 1941, to July, 1954, there were seventy patients admitted to the Minneapolis General Hospital with the established diagnosis of ectopic pregnancy. In 1942 Dr. William P. Sadler presented an analysis of the 102 cases of ectopic pregnancy admitted to Minneapolis General Hospital during the nine-year period, 1932 to 1940.

TABLE I. SEVENTY ECTOPIC PREGNANCIES. MINNEAPOLIS GENERAL HOSPITAL, JANUARY 1, 1941, THROUGH JULY 1, 1954

Age		Para		Race		Marital Status		Amenorrhea	
Under 20	5 patients	0	14	White	58	Married	50	None	2
20-25	12	1	21	Colored	10	Divorced	11	4 weeks	3
25-30	26	2	12	Indian	2	Single	6	5-8 weeks	27
30-35	17	3	10			Widowed	3	8-12 weeks	27
35-40	8	4	6					12 or more	9
40 and over	2	5	5					Unknown	2
		6	0						
		7	1						
		Unknown	1						

The current study analyzes the seventy cases from the standpoint of admission status, symptoms, and preoperative laboratory and physical findings; however, a more complete study, comparable to that of Dr. Sadler, is in the process of formulation.

The ages of these patients varied from seventeen to forty-two. Table I shows that the greatest number were in the 25- to 30-year age-group (twenty-six patients) with seventeen patients in the 30- to 35-year age-group. The racial distribution was: fifty-eight white patients, ten negroes and two Indians. Marital status was as follows: fifty married, eleven divorced, six single and three widowed. The parity, when known, varied from 0 to 7, with the largest number in the para 1 classification (twenty-one cases), and approximately equal distribution in the groups para 0, 2, and 3.

The record of amenorrhea was quite typical of ectopic pregnancy, with two patients reporting no amenorrhea, three with amenorrhea lasting four weeks, twenty-seven lasting five to eight weeks, twenty-seven lasting eight to twelve weeks, nine lasting twelve or more weeks, and two unknown.

The histories relative to vaginal bleeding prior to admission are shown in Table II. There was no bleeding in twelve cases, only one episode of bleeding in two cases, abnormal menses only in six cases, spotting only in seventeen cases, intermittent moderate bleeding in nine cases, continuous moderate bleeding in eighteen cases, profuse bleeding in seven cases, small clots passed in four cases, large clots passed in five cases, apparent tissue passed in six cases, and no history in one case.

TABLE II. VAGINAL BLEEDING AND PAIN PRIOR TO SURGERY

Bleeding		Pain	
None	12	None	1
One episode only	2	Shoulder pain	24
Abnormal menses only	6	Dull pelvic ache only	5
Spotting only	17	Abdominal cramping	18
Intermittent	9	Sharp pain with fainting	18
Continuous moderate	18	Sharp pain without fainting	41
Profuse	7	Rectal pain and or pain on defecation	17
Small clots	4	Dysuria	6
Large clots	5	Epigastric pain	5
Apparent tissue passed	6	Nausea and or vomiting	43
No history	1	No history	1

Type of pain experienced was also widely variable. The largest group, forty-one, reported sharp abdominal pain without syncope, and a substantial group, eighteen, reported sharp pain with syncope. Abdominal cramping alone or associated with

Presented before the Minneapolis Academy of Medicine, October 18, 1954.

other pain was reported by eighteen patients. Shoulder pain, in one or both shoulders, and always associated with some other type of pain, was reported by twenty-four patients. Nausea and/or vomiting, associated with pain or bleeding episodes, was reported by forty-three patients. was reported as soft and/or bluish in twenty-seven patients. The uterus, when palpable, was described as enlarged in twenty-one patients, and tender in nineteen patients.

Admission temperature was recorded in sixty-nine cases. It was found to be below 90 degrees F.

TABLE III. ECTOPIC PREGNANCIES ADMISSION STATUS

Temperature	Cases	Pulse Rate	Cases	Systolic Blood Pressure	Cases	Clinical Shock	Cases
Below 99° F	27	Below 80	8	Below 80 mm Hg	9	On admission	19
99-100°	34	80- 90	25	80- 90	5	One profound died 20 minutes after admission	
100-101°	7	90-100	9	90-100	2	Shock subsequent to admission	3
101-102°	1	100-110	8	100-110	10	No observation of clinical shock	48
Not recorded	1	110-120	2	110-120	12		
		120-above	10	120-130	17		
		Imperceptible	3	130-140	12		
		Not recorded	5	No record	3		

TABLE IV. ADMISSION BLOOD STUDIES

Hemoglobin	Cases	White Cell Count	Cases	Hemoglobin Drop	Cases
Below 40%	2	Less than		Less than	
40-50	8	9,000	18	5%	21
50-60	16	9-10,000	9	5-10	16
60-70	21	10-11,000	2	10-15	12
70-80	8	11-12,000	9	15-20	6
80-90	14	12-13,000	3	20-25	9
Not recorded	1	13-14,000	7	25-30	3
		14-15,000	3	30-35	0
Sedimentation rate done	26	Over 15,000	18	40%	2
Elevated	16	Unknown	1	Not recorded	1
Not elevated	10				

Rectal pain and/or pain on defecation reported by seventeen patients, is understandable when the findings of cul-de-sac changes are noted. A relatively small number of patients reported no pain, dull pelvic ache only, pain associated with urination, or epigastric pain.

Abdominal examination revealed tenderness of some kind in sixty-eight instances, rebound tenderness occurring in fifty-eight of these. Abdominal distention was present in fifteen instances, and rigidity in twenty-three instances. Abdominal mass or masses were recorded in eight instances. Pelvic examination revealed adnexal mass or masses in thirty-eight patients. In most of these reports, the masses are described as very tender. Adnexal tenderness without definite masses was reported in seventeen patients. In many patients, masses could not be outlined because of extreme tenderness. Cul-de-sac fullness or masses were described in thirty-four patients, and cul-de-sac tenderness without fullness in nine patients. Painful cervix was reported in forty patients, the most frequent description being extreme pain on movement of the cervix. The cervix in twenty-seven cases, 99 to 100 degrees in thirty-four cases, 100 to 101 degrees in seven cases, and 101 to 102 degrees in one case. The admission pulse rate, recorded for sixty-five patients, was below 80 in eight patients, 80 to 90 in twenty-five, 90 to 100 in nine, 100 to 110 in eight, 110 to 120 in twenty-two, above 120 in ten patients, and three patients were reported to have imperceptible pulses. Systolic blood pressure was recorded on admission in sixty-seven instances. The pressure was below 80 mm. Hg in nine instances, 80 to 90 in five instances, and 90 to 100 in two instances. The remainder of the admission readings varied from 100 to 140 mm. Hg. Clinical shock was noted on admission in nineteen patients; one patient, admitted in profound shock, expired twenty minutes later. Subsequent clinical shock developed in three patients who were not admitted in shock.

Admission hemoglobin determinations are reported in per cent since that is the method used at the hospital during most of the period covered by this study. Two patients had admission hemoglobin determinations below 40 per cent, eight had 40 to 50 per cent, sixteen had 50 to 60 per cent, twenty had 60 to 70 per cent, eight had 70 to 80 per cent, and fourteen had 80 to 90 per cent hemoglobin. Maximum hemoglobin drop was calculated. Twenty-one patients had less than 5 per cent drop, sixteen had 5 to 10 per cent, twelve had 10 to 15 per cent, six had 15 to 20 per cent, nine had 20 to 25 per cent, three had 25 to 30 per cent, and two had 40 per cent hemoglobin drop.

The admission white cell count varied widely. Eighteen patients had a count of 9,000 or less,

TABLE V. PREVIOUS PELVIC SURGERY AND PELVIC INFLAMMATORY DISEASE

Pelvic Surgery	Cases	Pelvic Inflammatory Disease	Cases
None	42	History only	1
Tubal ligation (Madelaner)	1	Findings only	22
Appendectomy	18	History and findings	19
Tubal pregnancy	6	No history or findings	28
Salpingectomy (indication?)	1		
Dilatation and curettage	4		
Uterine suspension	1		
Bartholins cyst	1		
Vaginal plastic	2		
Cesarean section	1		

TABLE VI.

Diagnostic Tests	Cases	Time Interval Admission to Surgery	Cases	Transfusion Prior or During Surgery	Cases
Posterior colpotomy or colpopuncture		Less than		None	28
Positive findings	20	3 hours	13	500 cc. or less	15
Equivocal	1	3- 5	8	500-1000	13
Negative	5	5- 7	5	1000-1500	4
Culdoscopy	1	7- 9	3	1500-2000	5
Freidman test		9-11	2	2000-2500	0
positive	19	12-24	8	2500-3000	4
		24-36	3	4000 cc.	1
		36-48 hours	1		
		48 hours to 16 days	26		
		One died prior to surgery	1		

TABLE VII. PHYSICAL FINDINGS

Abdominal		Pelvic	
Tenderness	68	Adnexal mass or masses	38
Distention	15	Adnexal tenderness without masses	17
Rebound	58	Cul-de-sac fullness or mass	34
Rigidity	23	Cul-de-sac tenderness without fullness	9
Abdominal mass or masses	8	Tender cervix	40
		Soft or changed cervix	27
		Tender uterus	19
		Enlarged uterus	21

nine patients had 9,000 to 10,000, two patients had 10,000 to 11,000, nine patients had 11,000 to 12,000, three patients had 12,000 to 13,000, seven patients had 13,000 to 14,000, three patients 14,000 to 15,000, and eighteen patients had a count of over 15,000. Factors influencing white cell count and hemoglobin were dehydration, blood loss and administration of intravenous fluids, plasma and blood.

Twenty-eight patients had had lower abdominal or pelvic surgery previous to the onset of the present illness. There had been one tubal ligation (Madelaner), eighteen appendectomies, four dilations and curettages, one uterine suspension, one excision of a Bartholin cyst, two vaginal plastic operations, one cesarean section, and six procedures for ectopic pregnancy, and one salpingectomy for reasons other than ectopic pregnancy.

History and/or evidence of pelvic inflammatory disease was found in forty-two patients. There were histories and operative findings of pelvic inflammatory disease in nineteen patients, operative findings only in twenty-two patients, and history but no operative findings in one patient. In some cases there was operative description of pelvic inflammatory disease without any pathological confirmation.

There were nineteen positive Friedman tests, and several cases in which the test results were not recorded. Posterior colpotomy or colpopuncture was positive in twenty cases, equivocal in one, and negative in five cases. Culdoscopy was done once.

Dilation and curettage was carried out on twelve patients. Decidual tissue was obtained six times, hyperplastic endometrium twice, proliferative endometrium twice, secretory endometrium once and insufficient tissue for diagnosis once.

The time interval from admission to surgery varied from less than one hour to sixteen days. The interval was less than 3 hours on thirteen occasions, 3 to 5 hours in eight cases, 5 to 7 hours in five cases, 7 to 9 hours in three cases, 9 to 11 hours in two cases, 12 to 24 hours in eight cases, 24 to 36 hours in three cases, 36 to 48 hours in one case, and 48 hours to 16 days in twenty-six cases. One patient expired twenty minutes after admission. Several patients were discharged and re-admitted prior to surgery.

Summary

1. A review of the symptoms and diagnostic findings of seventy ectopic pregnancies has been presented. This series is not to be considered as typical of ectopic pregnancy, but of ectopic pregnancy as encountered in indigent patients at the Minneapolis General Hospital.

2. The percentage of patients admitted in shock was almost identical in the two Minneapolis General Hospital series: 27.1 per cent in this series, and 27.4 per cent in Dr. Sadler's series.

3. Of these seventy ectopic pregnancies, 60 per cent (forty-two cases) were complicated by or associated with pelvic inflammatory disease in some manner. This particular phase of the study cannot be compared with Dr. Sadler's series because he included only those diagnoses of inflammatory disease verified by tissue study, whereas this study also includes diagnoses based on operative findings alone.

4. The high incidence of recurrent ectopics

(six cases) (8.6 per cent) may further illustrate the increased incidence of ectopic pregnancy in the inflammatory pelvis.

5. Study of the data on vaginal bleeding reveals wide variation with no predominant pattern; however, fifty-eight of the seventy patients (83 per cent) presented some type of abnormal bleeding associated with pain.

6. The high incidence of rebound tenderness, distention, cul-de-sac masses, adnexal masses and shoulder pain illustrates the marked pathologic processes in this series.

7. Admission hemoglobin levels and drops in hemoglobin were undoubtedly influenced by dehydration, blood loss and transfusions administered.

8. The long interval between admission and surgery in twenty-six cases illustrates the need for more rapid diagnosis. Culdoscopy might have shortened this interval, but this procedure must be undertaken with great care in the inflammatory pelvis.

Note: Final diagnosis recorded on these patients: ruptured tubal pregnancy, forty-eight; tubal pregnancy with probable rupture, nine; aborting or aborted tubal pregnancy, five; unruptured tubal pregnancy, seven; ectopic pregnancy, probable ovarian, one.

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OBSTRUCTION OF THE BOWEL IN THE NEWBORN

(Continued from Page 169)

revealed that distal loops of bowel beyond the ligament of Treitz were completely collapsed. There was meconium in the colon. The proximal duodenum was distended and the distal duodenum was collapsed. The diagnosis was annular pancreas. A retrocolic duodenal jejunostomy side-to-side anastomosis was performed. The child died November 11, 1950, twenty-two days after surgery. Autopsy revealed malformation of duodenum, pneumonitis, and necrosis of the brain. The anastomosis was functioning all right.

Summary

Six causes of bowel obstruction in the newborn, together with two case reports, have been presented. Various diagnostic points have been given. However, the important thing is not to differentiate the type of bowel obstruction as to etiology, but as to whether it is a complete obstruction, and whether it is a high or low bowel obstruction. The sooner this is done after birth, the better the prognosis for the child.

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Athletic Accident Benefit Plan, 1954-1955

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Minneapolis, Minnesota

IN JANUARY, 1952, a summary and explanation of the Athletic Accident Benefit Plan of the Minnesota State High School League, its limitations, administration, and methods of financing, was presented to the members of Hennepin County Medical Society in their *Bulletin*. At the time of this writing an up-to-date review with new revisions in this program is being announced to all of the member doctors of the Minnesota State Medical Association.

In spite of conscientious efforts by H. R. Peterson and by the State High School League, there resulted some controversies between Minnesota doctors and the league, mostly because of an honest lack of knowledge on the part of either the student athlete and his family or the doctor himself. There was a feeling by the parents that the payments by the league were *full* and satisfactory for medical care of the injury involved. There was an unfortunate misunderstanding by some doctors in the state that an attempt was being made by the state league to set up a fee-schedule for athletic injuries and that payments were too low. Fortunately huge strides have been made to eradicate both of these misunderstood aspects in the program.

The Medical Service Committee of the Minnesota State Medical Association has worked for several years in close co-operation with H. R. Peterson, executive secretary of the Minnesota State High School League, and feels that your committee is very close to a satisfactory solution. During this period, Mr. Peterson and his Board of Control have raised a great number of the benefits paid, especially on the common type of athletic injuries. They have devised a completely new method of perforated forms in order to attempt a more certain method of dispersing the information about the "benefit" plan, not insurance, to the student athlete and his parents. Printed on the Parents Permit Signature Card is the following statement "The undersigned further states that the 'Notice to Athletes and Parents' has been received; and understands the Benefit Plan provides only limited financial assistance; and the balance of any bill, if not paid in full, is a responsibility of the pupil or his parents."

The extremely controversial paragraph in the old explanatory bulletin read, "The plan provides limited coverage only: The schedule of maximum allowances *grants reasonable payments for normal services.*" This has been removed from the new forms and only the following paragraph used: "One of the major services provided by the League is the Athletic Benefit Plan which offers financial assistance in meeting the cost of medical, dental, and hospital services resulting from injuries incurred while participating in athletic activities sponsored by the school. The plan is a non-profit mutual benefit program and not insurance. It provides specific limited allowances for various injuries, and the balance of the bills, if not paid in full, is a responsibility of the athlete or his parents". Your committee feels that this should do much to clarify the apparent misunderstanding between the league and the doctors of medicine in the State of Minnesota.

The financing of this plan is done by charging in the past $2 per year to the boy who participates in football and other sports, and $1 per year if he does not participate in football. This fee was known to be insufficient for sound underwriting but many boys in the state from some of the less fortunate families have been unable in the past to afford even this fee. The deficit of the underwriting was made up from profits of the Minnesota State High School Basketball Tournament each year. Fortunately for the success of the plan, the weather and competition have co-operated to make a financially successful basketball tournament. However, realizing that this was not sound and there could be "many a slip 'twixt the cup and the lip", the Board of

(Continued on Page 214)

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Seminar

DIAMOX

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Diamox (2-acetylamino-1, 3, 4 thiadiazole-5-sulfonamide) is a heterocyclic sulfonamide which was developed by the American Cyanamide Company in the search for a powerful carbonic anhydrase inhibitor related to sulfanilamide. In 1940, Mann and Keilin[17] discovered that sulfanilamide and other compounds with a free sulfonamide group inhibit carbonic anhydrase activity. An amino group in the para position was found necessary for the antibacterial action but was not necessary for the inhibitory effect on carbonic anhydrase (Fig. 1). Following the observation that sulfanilamide induced acidosis with an alkaline urine,[21] Höber[14] suggested that alkalization of the urine was due to inhibition of carbonic anhydrase in the renal tubules. Subsequently, Pitts and Alexander[24] showed that sulfanilamide reduced the capacity of the kidney to eliminate acid under maximal stimulation by a phosphate buffer load.

In 1949, Schwartz[26] administered sulfanilamide to three patients with congestive heart failure and observed an increased excretion of sodium, potassium, and water. Although clinical improvement and reduction of edema was noted, the drug was considered to possess too little carbonic anhydrase activity and to be too toxic for continued use.

Chemistry and Pharmacology

Diamox is 40 to 500 times as potent a carbonic anhydrase inhibitor as sulfanilamide. The pharmacological properties have been described by Maren and co-workers.[18,19,20] (Fig. 1.)

Diamox is well absorbed from the gastrointestinal tract in both the experimental animal and man. It possesses very low systemic toxicity and is excreted largely unchanged by the kidney in twenty-four hours. The renal clearance is approximately two-thirds that of the glomerular filtration rate in the dog. Studies with Diamox and carbonic anhydrase both *in vivo* and *in vitro*, show that the interaction between inhibitor and enzyme is reversible.

Following a single oral dose, there is increased urinary excretion of sodium, potassium, bicarbonate and water. The pH of the urine increases and titratable acid and ammonia excretion are sharply reduced or abolished. This effect lasts

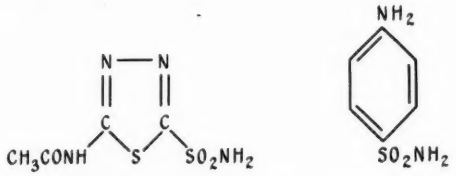

Fig. 1.

about six hours and then reverses with the onset of the recovery phase. This phase lasts about eighteen hours and is marked by urine almost free of bicarbonate and sodium, low in potassium, and with increased titratable acid and ammonia. There is a drop in plasma bicarbonate and pH during the diuretic phase which returns to normal during the recovery period. This effect in the rat is largely independent of the dose in the range of 10-1000 mg./kilo. Dogs given 100 mg./kilo/day for sixteen months showed an initial renal response but then the urinary pattern reverted to normal after the first few days although there was a sustained acidosis characterized by a low blood pH and plasma bicarbonate and elevated chloride. The serum sodium and potassium remained normal. Urinary and plasma electrolytes reverted to normal in three to four days following withdrawal of the drug. If the dog was then challenged with Diamox, the response was the same as with initial treatment. These results were in contrast to those in dogs maintained on 5 to 10 mg./kilo/day where there was a continued renal effect and little or no acidosis. On low daily doses extra base output was effectively countered by extra base conservation during the recovery periods. On high or multiple doses the animal became refractory to the diuretic effect and remained acidotic. The drug is not cumulative with long-term administration and has no preference for any tissue, except red cells, which it appears to saturate at a fairly low level.

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Presented at Medical Grand Rounds, Veterans Administration Hospital, Minneapolis, Dr. E. B. Flink, Chief of Medical Service.

Tomashefski et al[30] and Carter[8] have found after the intravenous administration of Diamox to dogs that the alveolar P_{CO_2} was significantly less than the arterial P_{CO_2}. Shepard et al[28] reported

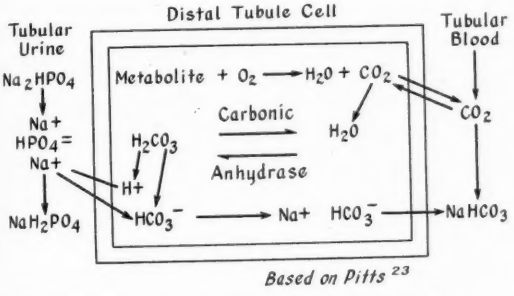

Fig. 2. Process of acidification by renal tubule.

similar findings in two normal human subjects after exercise. These findings are thought to result from interference with the release of carbon dioxide from pulmonary capillary blood after inhibition of carbonic anhydrase.

Diamox exerts a marked inhibitory effect on pancreatic secretion in the dog following stimulation with secretin.[5] It has also been shown both in dogs[15] and ulcer patients[29] that Diamox causes a significant inhibitory effect on hydrochloric acid production.

Mechanism of Action

In order to understand better the renal action of Diamox, a brief discussion of acid-base balance by the kidney will be presented. Two hundred liters of plasma are filtered through the glomeruli each day. Each liter of plasma contains approximately 25 mM. of bicarbonate. The total bicarbonate filtered is 5,000 mM., which is five times the body store of bicarbonate.

Obviously the bicarbonate must be filtered over and over again and reabsorbed each time by the renal tubules as only 1 to 2 mM. escapes into the urine each day. Sulfuric and phosphoric acids are formed in considerable quantities in the metabolism of phospholipids and proteins. The excretion of titratable acid and ammonia reverse the process of neutralization and promote the elimination of acid anions minus the base. These two renal mechanisms prevent exhaustion of the bicarbonate stores.

Carbonic anhydrase accelerates the reaction of carbon dioxide and water to form carbonic acid in the tubule cells. The ionization of carbonic acid to form hydrogen and bicarbonate ions provides hydrogen ions which are exchanged for sodium ions of the buffers of the glomerular filtrate, chiefly in disodium phosphate (Na_2HPO_4) and sodium bicarbonate ($NaHCO_3$). The excretion of sodium acid phosphate (Na_2HPO_4) in the urine conserves sodium which returns to the blood. (Fig. 2).

Ammonia (NH_3) is formed from glutamine

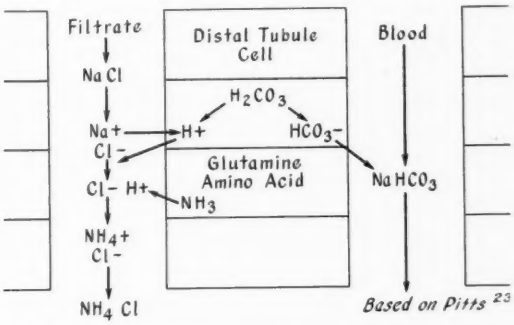

Fig. 3. Process of ammonia production by the renal tubule.

and other amino acids in the distal tubule cell. Each molecule of ammonia secreted into the tubular urine binds one mole of hydrogen and permits one mole of sodium to be reabsorbed. Thus ammonia is exchanged for base, meq. for meq., although the exchange is indirect (Fig. 3). The rate of ammonia excretion is inversely proportional to the pH of the urine; i.e., the more acid the urine, the more ammonia is excreted, and vice versa.

Inhibition of carbonic anhydrase in the kidney leads to the following:

1. Decreased urine acidity due to decreased availability of hydrogen ion from carbonic acid.

2. Increased urinary excretion of sodium due to decreased sodium reabsorption.

3. Increased urinary excretion of potassium due to increased tubular secretion of potassium. Berliner[3] has shown that potassium ions compete with hydrogen ions in the tubules for excretion; hence decreased hydrogen ion secretion leads to an increased potassium output.

4. Decreased ammonium excretion due to limited availability of hydrogen ion for combination with ammonia. This also leads to increased excretion of sodium.

5. Increased total carbon dioxide content of the urine due to increased excretion of bicarbonate. Normally, hydrogen ion secreted by the renal tubule cell reacts with bicarbonate in the tubular urine and the carbon dioxide evolved diffuses back into the blood. When hydrogen ion secretion is blocked by carbonic anhydrase inhibitor this reaction does not take place and bicarbonate excretion is therefore increased.

Clinical Applications of Diamox

Friedberg, Halpern and Taymor[12] studied the intravenous effect of Diamox on twelve hospitalized and ambulatory patients with congestive heart failure of varied etiology and on three patients without evidence of cardiovascular or renal disease. All of the cardiac patients had been treated in the previous few weeks and had little evidence of edema except one patient who had moderate edema of the lower extremities. A single intravenous injection of 0.75 gm. of Diamox was given. Urinary output increased in each case, and the urine contained increased amounts of sodium, potassium, and bicarbonate. Chloride and phosphate excretion were only slightly increased and appeared to be related to the increased urinary flow. The urine became alkaline about thirty minutes after injection and remained so for a period of about six hours. Titratable acid rapidly disappeared. Urinary ammonium was markedly diminished in all cases; however, it continued to be present even when the urine was more alkaline than the plasma. The sodium excretion increased markedly in every instance independent of the presence or absence of congestive heart failure. In two of the control subjects who received 5 to 7 grams of salt a day, the sodium excretions increased four to six times over the control values. In the other control subject on a 500 mgm. sodium diet, there was a tenfold increase in sodium excretion over the control value. No significant effect was noted in plasma concentration of sodium, potassium, chloride, phosphate or creatinine. There was a slight and inconstant tendency towards a mild acidosis as indicated by a decrease in plasma bicarbonate level and blood pH.

Leaf, Schwartz, and Relman[16] performed balance studies on five patients with congestive failure and on one normal subject before and after the oral administration of Diamox in varying dosage schedules. Two of the patients with congestive heart failure and the normal subject demonstrated the usual responses. The other three patients lost no sodium and responded only with increased potassium excretion and reduction in urine acidity. Unresponsiveness to the drug developed rapidly even with intervals of one to two days between doses, and in one case appeared after a single dose of 0.25 gm. They noted that the onset of drug resistance was always associated with the development of hyperchloremic acidosis.

Friedberg et al[13] studied the oral effect of Diamox in twenty-six patients with various types of heart disease and found an increased excretion of sodium and potassium in the urine of all patients regardless of clinical response. There was slight to moderate increase in chloride excretion in sixteen patients and a decrease in eight. Plasma levels of sodium, potassium, and chloride were not altered significantly. The carbon dioxide content fell in most cases, and in some was accompanied by a slight fall in the blood pH. "Excellent" or "good" clinical responses were claimed in eighteen of the twenty-six patients although the mean weight loss was only 4.4 pounds in the hospital group and 3.8 pounds in the clinic patients. Several dosage schedules were employed, but 0.25 gm. every eight hours for two days and repeated every two to seven days or the daily administration of 0.25 gm. were the most effective. Patients who did not respond to smaller doses did not have a diuresis with larger doses nor did prolongation of the course more than two days produce a diuresis in these patients. In the patients who responded satisfactorily to Diamox, diuresis and weight loss continued until "dry weight" was reached which could be maintained as long as the drug was continued.

Relman, Leaf, and Schwartz[25] reported disappointing results following the oral administration of Diamox in twenty-six hospitalized patients with severe congestive heart failure. Approximately half of the patients failed to lose weight and only one of the remainder became edema free, while the others lost 2 to 6 pounds over a seven-day period. All but three patients had a definite pharmacological effect from the drug even when there was little or no diuretic response. Twelve patients had blood nonprotein nitrogen concentrations of 50-100 mgm. per 100 cc. and in fourteen the level was below 50 mgm. per 100 cc. Only three of thirteen courses of treatment in azotemic patients resulted in weight losses of 4 pounds or more; however, seven of fifteen patients without azotemia had this same response. This suggested to the authors the importance of renal function. The explanation of this response is not clear, but it was postulated that carbonic anhydrase is less important for the over-all reabsorption of bicarbonate when the glomerular filtration rate is low and the filtration of bicarbonate is reduced. Another possibility considered was that the azotemia was simply a reflection of severe circulatory insufficiency and therefore of the factors operating to retain sodium. In spite of the disappointing results the authors refer to some encouraging studies[27] in the treatment of congestive heart failure due to chronic cor pulmonale where Diamox appeared to effect large diuresis and significant clinical improvement.

Belsky[2] reported a definite diuresis in all of thirteen patients with congestive heart failure treated with oral Diamox. Eleven of the thirteen patients were kept edema-free with more or less constant weight on a daily dose of 0.5 gm. of Diamox, low salt diet and digitalis. He noted that Diamox alone or in combination with mercurial diuretics had little diuretic effect in the presence of anascarca or renal decompensation in two patients. However, after removal of ascitic fluid, the effectiveness of Diamox appeared to be increased, although supplementary mercurial injections were required.

Nadell[22] reported on the effect of Diamox in two patients with respiratory acidosis and two normal subjects. In three of four patients, the arterial pH fell by 0.1 to 0.2 pH units. After three days, the urine pH and electrolyte values returned to control values and remained unchanged even when the drugs were given for periods of twenty-five to fifty-five days. However, during the entire period the arterial carbon dioxide was decreased 5-10 mEq/l. The control patients remained asymptomatic during the entire period. One of the two patients with respiratory acidosis continued to be alert outside of the respirator on two separate occasions while getting Diamox. When the drug was discontinued, a more marked respiratory acidosis developed. The other patient developed marked drowsiness fourteen days after therapy was begun. This cleared when the dosage was decreased from 10 to 5 mgm. per kilo.

Fishman, Samet, and Cournand[11] found a diminished ventilatory response following an increase in inspired carbon dioxide in patients with pulmonary emphysema with carbon dioxide retention as compared with emphysematous patients without carbon dioxide retention and with normals. The emphysema patients with carbon dioxide retention were given Diamox over a fifteen-month period. Although the arterial P_{CO_2} and alkali reserve returned to normal there was no improvement in the ventilatory response to carbon dioxide.

Bergstrom[4] et al reported on the effect of Diamox in forty-two epileptic persons with intractable seizures. The rationale underlying such therapy is the production of a mild metabolic acidosis similar to that produced by ketogenic diets. Ninety to one hundred per cent seizure control was obtained in four instances and 50 to 90 per cent in four others. None was made worse. The authors concluded that Diamox is useful in only a small number of cases refractory to other measures.

Becker[1] treated nineteen patients with glaucoma with single doses of 0.5 to 1.0 gms. of Diamox. In all cases, the intraocular pressure began to decrease in 60 to 90 minutes. It reached a minimum in three to five hours and returned to initial levels in eight to twelve hours. Because of the high concentration of bicarbonate in the posterior chamber it was postulated that carbonic anhydrase plays an essential rôle in the formation of aqueous humor. It was not determined whether its mode of action is to inhibit the secretory mechanism directly or whether the fall in intraocular pressure is secondary to induced systemic electrolyte alteration.

Diamox has been tried in the treatment of several conditions associated with edema formation. The impaired renal function in chronic glomerulonephritis probably interferes with the effectiveness of Diamox as a diuretic. However, since it had not proven dangerous, it was thought to be worthy of a trial.[9] Equivocal results have been obtained in the treatment of cirrhosis.[6,7]

Toxicity

No serious toxic effects have been reported with Diamox. Paresthesias of the face and hands and drowsiness have been observed frequently and appear to be related to the dosage.[2,4,13] These side effects have been encountered when the daily dose of Diamox was 1.0 gm. or more and were reduced to a minimum or disappeared when the dose was decreased to 0.5 or 0.25 gm. per day. Bergstrom et al[4] reported fatigue, flushing, polydipsia, hypernea, paresthesias, and headache with doses of 10 to 30 mg. per kilo in the treatment of epileptics but did not state the number of cases involved. A few instances of disorientation and irrational behavior have been observed in cirrhotic patients treated with Diamox.[10] No instances of renal damage, hematologic disturbances* or skin manifestations have been reported.

Summary

Diamox is a potent carbonic anhydrase inhibitor affecting carbonic anhydrase contained in red cells, kidney, stomach, pancreas, and eye. The usual pharmacological response is characterized by the diuresis of an alkaline urine containing increased amounts of sodium, potassium, and bicarbonate and a decreased plasma bicarbonate and blood pH.

The results of Diamox administration in various disease states have been reviewed. The drug is effective both orally and via the intravenous route. The toxicity is low and consists mainly of paresthesias and drowsiness.

Further clinical trial appears warranted.

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GYNECOLOGICAL CANCER DETECTION

(Continued from Page 178)

depend on detection of carcinoma at a stage amenable to treatment. The accessibility of the female pelvic organs makes this region nearly ideal for the early detection of malignancy.

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Laboratory Aids

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George G. Stilwell, Editor

LIMITATIONS OF THE WIDAL TEST

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THERE are three reasons why it is necessary to re-evaluate the Widal test as a laboratory aid in the diagnosis of typhoid fever: (1) knowledge regarding the antigenic relationship of Salmonella typhosa (Eberthella typhosa) to many other Salmonella organisms; (2) the Widal test is essentially of no diagnostic value in the increasingly large number of persons who have had inoculation with typhoid and paratyphoid A and B vaccine; (3) the incidence of typhoid infection in Minnesota is low.

Typhoid O (somatic) and typhoid H (flagellar) antigens are used in the quantitative Widal test. There are approximately 63 Salmonella organisms that possess one or more of the somatic antigens found in S. typhosa. Accordingly, a person who may be infected with and produce antibodies to any one of these organisms gives a satisfactory titer when his serum is tested with O antigen. For example, we have not infrequently demonstrated O antibody titers as high as 1:1,280 in serum from patients with enteric infection and have isolated only S. enteriditis from the stools of these patients. The high O antibody titer in these patients can be easily explained since S. enteriditis possesses two somatic antigens identical to those of S. typhosa.

It is frequently stated that during the early stages of typhoid infection the O titer is higher than the H titer and that a high O titer is diagnostic of typhoid fever. We cannot agree with this statement. The high O antibody titer may be due to an organism with somatic antigens identical to the somatic antigens of the typhoid bacillus. We found that H titers in the great majority of cases were higher than O titers in a study of agglutination reactions in serum from 103 persons reported to be ill with typhoid fever. Also negative results were found in 5.8 per cent of the O tests in this study as compared to negative results in only 1.0 per cent of the H tests. In a study of the agglutination pattern of forty-seven ill patients diagnosed as having typhoid fever and from whom S. typhosa was isolated, the H antibody titer in the great majority of patients was higher than the O titer; in two patients there was no demonstrable O titer, only H agglutinins being detectable.

Thus it would appear that perhaps the H antibody titer would offer more certain aid in the diagnosis of typhoid fever. A study of the H and O antibody titers on serum from 8,945 ill persons who did not have typhoid fever showed that the H antibody titer was consistently higher than the O titer. This was particularly true of serum obtained from 787 persons who had received typhoid vaccine. In this vaccinated group, the results in 43 per cent of the H agglutination tests were positive in dilutions of 1:320 or more, whereas only 2.4 per cent of the O agglutination titers fell in this range. Negative results of H agglutination tests were observed in 24.1 per cent of this vaccinated group as compared to negative results in 55.6 per cent of the O agglutination tests. In a study of serum from 8,075 ill persons who did not have typhoid fever and who gave no history of having had typhoid fever or vaccination, 3.3 per cent of the H agglutination tests gave positive results in dilutions of 1:320 or more, whereas only 0.3 per cent of the O agglutination tests were in this range. Thus it is obvious that we cannot depend on H agglutination as an aid in the diagnosis of typhoid fever.

Perhaps it may be argued that differences in the H and O antibody titers would help in making a diagnosis. Unfortunately no definite single H and O titer is diagnostic of typhoid

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This is the twenty-fifth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

Dr. Bauer is Director, Division of Medical Laboratories, Minnesota Department of Health.

fever. The higher the titer chosen for the diagnostic dividing line, the more likely is such titer to indicate infection but at the same time the smaller will be the number of cases of typhoid thus diagnosed.

We may try to get some value out of the Widal test as a diagnostic aid by testing serial specimens of blood for H and O antibodies. Again we are frustrated by persons who may have had typhoid fever and by the great many people who have received typhoid vaccine. No diagnostic value can be placed on the H agglutinins in a vaccinated person or a person with a history of previous typhoid fever. Even a considerable increase in titer is of little or no diagnostic importance since this may be due to nonspecific causes. No reliance can be placed on O agglutinins since other organisms share common O antigens with S. typhosa. A blood culture done early in the disease would give specific results and in a shorter period than would Widal tests done on a series of specimens of blood.

Since the Widal test is of questionable value in the specific diagnosis of typhoid fever and of no diagnostic value in vaccinated or previously infected persons, we might ask if it is of value in the detection of typhoid carriers. Serologic studies were done on thirty-seven bacteriologically proved typhoid carriers. Nineteen of these carriers (51 per cent) were negative when tested with O antigen including seven who were also negative when tested with H antigen. Eighteen (49 per cent) of the thirty-seven carriers tested showed both H and O antibodies, but the H antibody titers were higher. Although H agglutinins were present more frequently and in higher titer than were O agglutinins in these proved carriers, the same was found to be true in examination of serum from 229 well persons who gave a history of typhoid vaccination, from twenty-eight persons who gave a history of previous typhoid infection and from 2,355 persons who gave no history of vaccination.

Conclusions

1. No arbitrary titer of either H or O agglutinins can be accepted as diagnostic for typhoid fever.

2. Since Salmonella typhosa shares common O antigens with other Salmonella organisms, the O agglutination test is not specific for typhoid.

3. The Widal test is of no practical value in the diagnosis of typhoid fever in vaccinated persons or those with a history of previous typhoid fever.

Recommendations

1. If typhoid or paratyphoid infection is suspected, culture of blood and stools should be done early in the illness. Blood for culture should be collected during the first two weeks of illness. Examination of stools is the procedure of choice after the first two weeks of illness.

2. Stools should be submitted for culture from all persons having enteric infection suggestive of salmonellosis or shigellosis.

3. Since Salmonella typhosa must be isolated from suspects to prove their carrier status, it is thought that examination of stools from the following persons will yield more productive results than will the Widal test: (1) those giving a history of typhoid fever; (2) those who have resided with or prepared food for any persons who have contracted typhoid fever; (3) those who have symptoms suggestive of gallbladder disease; (4) food handlers who, in addition to any of the conditions just mentioned, may give a history of a recent intestinal upset of any nature.

REVIEWS OF MEDICAL MOTION PICTURES

The Committee on Medical Motion Pictures of the American Medical Association announces that Booklet 6 of Reviews of Medical Motion Pictures is now ready for distribution. This booklet contains sixty-four critical reviews of medical and health films which were published in *The Journal* during 1954.

A copy has been sent to the secretary of each state medical society and the books are available to county medical societies from the Committee on Medical Motion Pictures. Other requests should be sent to the Order Department, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.

The price of individual booklets is 25 cents each or the complete set of six booklets including all reviews published since 1946 is available for $1.00.

Clinical-Pathological Conference

Sponsored by
The Minnesota Society
of Clinical Pathologists
Donald F. Gleason, Editor

CASE PRESENTATION

DONALD F. GLEASON, M.D.
Minneapolis, Minnesota

Clinical History

A fifty-three-year-old bartender was first admitted to this hospital for treatment of seborrheic dermatitis and for injection of varicose veins, January 30, 1942. He stated that for six weeks he had been receiving intravenous and intramuscular injections, but did not know for what purpose. His blood pressure was 132 mm. Hg systolic and 80 mm. diastolic. Urinalysis showed no abnormalities. The blood Wassermann test resulted in negative findings.

On April 14, 1951, the patient, now aged sixty-two, was admitted to the hospital complaining of epigastric pain for three weeks. Five weeks prior to admission, he had been awakened by a severe constrictive retrosternal pain which extended into his left arm. He was hospitalized elsewhere for two weeks at bed rest. Thereafter, he had no appetite and had suffered three or four attacks of severe colicky pain in the epigastrium which extended across the right upper abdomen and to the right scapular area. He believed that these attacks were caused by eating fried or greasy foods. At times, he had nausea and vomiting during the attack. There had been no chills, fever, jaundice or darkening of the urine.

For three years, he had experienced substernal pain extending into the left arm on exertion, promptly relieved by rest or nitroglycerin. For a year, he had some dyspnea on exertion but no orthopnea nor paroxysmal nocturnal dyspnea. He had had gonorrhea forty years ago but denied having had syphilis. He had been drinking alcoholic beverages excessively for several years.

His mother had died of "heart trouble" at fifty-eight years, his father had died of a "stroke" at fifty-eight years, and one brother had died of "heart trouble" at forty-two years.

The patient was well developed, well nourished and in no acute distress. The pupils reacted normally. The temperature was 98.6 degrees F., the pulse rate 84 per minute. The lung fields were normal to percussion and auscultation. The heart sounds were of good quality and no murmur was heard. The aortic second sound was louder than the pulmonary sound. The point of maximal impulse was in the fifth interspace at the anterior axillary line. The blood pressure was 170 mm. Hg systolic and 116 mm. diastolic. The abdomen was normal apart from moderate epigastric tenderness. Neurological examination showed no abnormalities.

The hemoglobin was 11.3 grams per 100 ml., the leukocyte count 7,250 per cu. mm. (65 per cent neutrophils, 33 per cent lymphocytes and 2 per cent eosinophils), the erythrocyte sedimentation rate 60 mm. at one hour. Specific gravity of the urine was 1.014, the sediment contained 0 to 3 erythrocytes and 0 to 1 leukocyte per high power field, the test for albumin was 1 plus. A catheterized urine specimen showed 15 per cent excretion of phenolsulphonphthalein in one-half hour. The blood Wassermann test was negative, the Kahn 3 plus, the Hinton 4 plus, the VDRL slide test positive in 1 to 2 dilution. Examination of the spinal fluid on two occasions was completely negative, including total protein, complement fixation and gold curve. There was 7.2 per cent retention of bromsulfalein at forty-five minutes, the cephalin flocculation test gave negative findings, the thymol turbidity 5.2 units, the serum bilirubin 0.1 mg. per 100 ml. at one minute and a total of 0.5 mg. A two-hour urine urobilinogen was within normal limits. The blood urea nitrogen ranged between 16 and 25 mg. per 100 ml., the total protein was 6.9 gm. per 100 cc. (3.9 albumin and 3.0 globulin). Five stools showed no occult blood. The serum amylase was 56 units.

The posteroanterior radiograph of the chest showed a left ventricular cardiac contour, marked dilatation of the ascending aorta, tortuosity of the descending aorta and fluoroscopy confirmed the presence of calcification within the ascending aorta. An upper gastrointestinal series and cholecystogram were negative. Barium enema showed numerous diverticula in the lower colon. Radiographs of the skull showed calcification in the vessels of the circle of Willis. An electroencephalogram showed abnormalities, but with no specific connotation. Electrocardiograms showed left axis deviation, sagging ST and negative T in V5 and V6.

Shortly after admission, the patient experienced an episode lasting for two hours, characterized by numbness and weakness of the left arm. There was definite transient weakness of the left forearm during that time, but no abnormal neurological signs were demonstrated either during or after this episode. Throughout his course, he complained constantly of a severe, generalized headache which was not relieved by codeine. There was anorexia and occasional nausea and vomiting not described as projectile. Within five weeks, he had lost 12 pounds in weight. On the morning of June 24, he was found to be comatose, gasping, cyanotic and promptly expired.

Discussion

DR. SMITH: For three years, this man, now sixty-two, experienced substernal pain which extended into the left

From the Minneapolis Veterans Hospital; Dr. Gleason, pathologist.

arm on exertion and which was promptly relieved by rest or nitroglycerine. For a year, he had some dyspnea on exertion but neither orthopnea nor paroxysmal nocturnal dyspnea. This, of course, is the classical description of angina pectoris. To me, it means that the man has myocardial ischemia. There is a host of diseases which will cause myocardial ischemia, the most important of which is coronary artery disease, and it is by far the most common cause in our "macro-molecular" human population.

Five weeks prior to admission, he had been awakened by a severe constructive retrosternal pain which extended into his left arm. He was hospitalized elsewhere for two weeks' bed rest. Myocardial ischemia occurring during sleep has a different connotation. Ischemia can occur during sleep if an individual has frightening nightmares or if for some reason a hypertensive patient drops his blood pressure to low levels. Most often, it is due to an increment of coronary artery disease, usually thrombosis. If this ischemia is lasting, usually over fifteen to thirty minutes, some myocardial necrosis will occur, and we clinically call it infarction. I assume that the diagnosis of myocardial infarction was made when this patient was hospitalized elsewhere.

DR. SAMUEL NESBITT: That diagnosis was made.

DR. SMITH: The protocol states that he had gonorrhea four years ago but he denied having syphilis. That is a very circumstantial bit of evidence for a diagnosis of syphilis, but he stated he had been treated for six weeks with intravenous and intramuscular injections, although he did not know their purpose. In 1942, the treatment in vogue, of course, was combined mapharsen and bismuth therapy. It would have been possible to confirm this treatment with an x-ray of the gluteal region to look for intramuscular deposition of bismuth. Possibly even now, the radiologist can aid in this problem. Dr. Nesbitt used the past perfect tense here, "he had been receiving." I assume that this was just a short period before admission, and the serological test for syphilis was presumably negative when seen by his physician then. This causes me to wonder what evidence his physician had in 1942 to start this therapy.

Ordinarily, physical examination will help us somewhat in the differential diagnosis of the cause of angina pectoris. There was no evidence of any valvular disease. He did have hypertension and, of course, hypertension tends to accelerate atherosclerosis. The blood Wassermann test gave negative findings, the Kahn was 3 plus, the Hinton 4 plus and the VDRL slide test positive in 1:2 dilution. We have already some presumptive evidence that this man had syphilis and I will add this as very strong evidence. In late syphilis, this discrepancy is seen fairly commonly—complement fixation tests being negative while macro and micro flocculation tests are positive. If we were seeing this patient today, perhaps treponema immobilization tests would be of value.

The posteroanterior radiograph of the chest showed a left ventricular cardiac contour. There is a discrepancy between the radiograph as just described and the physical examination. I will accept the radiograph in this controversy. I think that physical examination of the heart is a very difficult and sometimes confusing procedure. Further, the radiographic examination showed marked dilatation of the ascending aorta, tortuosity of the descending aorta, and fluoroscopy confirmed the presence of calcification within the ascending aorta. This latter finding I would like to call pathognomonic evidence of lues. It is probably better evidence than a pathologist's examination. A study reported in 1945 by Jackman and Lubert[2] showed that in patients studied at post mortem, in whom the diagnosis of syphilis was possible pathologically, 22 per cent had calcification of the ascending aorta. Two patients with a pathological diagnosis of only arteriosclerosis had similar calcification. In one of these, there was serologic but no pathologic evidence of syphilis. The other patient had very diffuse and extremely severe arteriosclerosis. I think that this is firm evidence. We could pause here and let Dr. Olson show us the radiograph.

DR. P. A. OLSON: The chest radiograph shows the important features which Dr. Smith discussed. There is very distinct linear density in the wall of the ascending aorta which is also dilated to aneurysmal degree. It is sometimes hard to state what is aneurysmal and what is just dilated, but certainly this is wide enough so we would call it aneurysmal. There is calcification in the ascending aorta, so we should certainly consider that lues is present until proven otherwise. The heart as seen in the postercanterior view shows moderately severe enlargement of the left ventricle. The second shadow, the denser one medial to the left heart border, is the descending aorta. We cannot visualize its right border. Whether he has another aneurysm here is difficult to determine, but the aorta is certainly wider than it should be and is rather tortuous. The gastrointestinal series is normal, save for a diverticulum of the duodenum. This one radiograph of the gall bladder shows that the dye is not concentrated well, but there is no evidence of calculi.

DR. SMITH: The electrocardiogram showed left axis deviation, sagging ST and a negative T in V 5 and V 6. In addition, he has very low R waves across the anterior precordium. I would interpret these changes as subendocardial damage which might have well extended transmurally, anteriorly.

Now, I would like to go back to the reason for his admission. His presenting complaints were in the gastrointestinal system. Examination of the abdomen revealed no abnormalities except for moderate epigastric tenderness. His leukocyte count was normal and the erythrocyte sedimentation rate was 60 mm. There was a 7.2 per cent retention of BSP. The serum amylase was 56 units—I wonder if that was drawn at a time when he was having distress.

DR. NESBITT: We do not know.

DR. SMITH: We could see those abdominal x-rays now.

DR. OLSON: We can see marked calcification in this area which is typical of calcified abdominal aorta. The barium enema shows only a few diverticula in the sigmoid and the lower descending colon. There are moderately severe arthritic changes. There is no evidence of bismuth in the buttocks.

DR. SMITH: This brings up the problem of gastrointestinal complaints in patients with heart disease—a discussion about abdominal angina. In a man with a very compromised coronary blood flow, the mere addition of heavy meal may cause distress. One other possibility which has been advanced more frequently recently is that people who have diffuse vascular disease actually have mesenteric artery insufficiency and distress on this basis.

I am unable at this stage to make a diagnosis of hepatic cirrhosis in this man. I do not think there is any strong evidence of intrinsic gastrointestinal disease. Specific gravity of the urine was 1014. He had a 1 plus albumin and 15 per cent excretion of phenolsulphonphthalein in one-half hour. This depression, along with a normal blood urea nitrogen value, is quite a common functional derangement in patients with hypertension with efferent arteriolar constriction.

So now we go to the next complaint. His neurological examination was normal at the time of admission.

He had two spinal fluid examinations completely negative. Did this include dynamics?

DR. NESBITT: Yes.

DR. SMITH: A radiograph of the skull showed calcification in the vessels of the circle of Willis. Shortly after admission, according to the protocol, the patient experienced an episode lasting for two hours characterized by numbness and weakness of the left arm. "There was definite transient weakness of the left forearm but no abnormal neurological signs." I think these two statements are mutually exclusive. If he had true weakness, it would have to be listed as neurological, unless it is known to be muscular. I assume it is meant that he had no reflex changes. Throughout his course, he complained constantly of severe generalized headache which was not relieved by codeine. He had vomiting, not described as projectile. He lost 12 pounds in weight. Finally, he was found to be comatose, gasping, cyanotic and promptly expired. Do we know how long before he was found to be comatose that he was last observed?

DR. NESBITT: He was seen about an hour before. He was found in distress and he died in a very few minutes after that.

DR. SMITH: This finding of calcification in the vessels of the circle of Willis in a man who has hypertension, is not really very helpful. Camp[2] states that 4 per cent of any given group of people of age sixty would have such calcification. I would like to see those x-rays now.

DR. OLSON: Maybe you can't see it, even from the front row, but there is calcification in the internal carotid artery. There are several other little linear calcified densities in the region of the sella, the exact site of which is not definite but appears to be in vessels. The internal carotid is definitely calcified. The pineal gland is not calcified, so we cannot use it as a landmark as far as shift is concerned. No other abnormality is seen.

DR. SMITH: Well, we have a man with angina pectoris, myocardial ischemia, and luetic aortitis. The problem now is, why does he have coronary artery insufficiency? He did not have aortic insufficiency. Most patients who have ostial involvement of the coronaries have aortic insufficiency. Even when aortic insufficiency is present most patients who have evidence of myocardial ischemia do have coronary artery disease. We have evidence that he has arterial disease elsewhere. I would prefer to think that he has coronary atherosclerosis in addition to his luetic aortitis. He has hypertension, the cause of which we do not know. We know that he was normotensive at the age of fifty-three and that he became rather severely hypertensive thereafter. We do have evidence that this vascular disease is rather far advanced and that he had cardiomegaly otherwise poorly explained; I would prefer to think that his hypertension was of relatively long standing and its cause will not be disclosed. In the kidneys, he should have changes of benign hypertension with or without arteriosclerosis. I mentioned the gastro-intestinal complaints and the fact that I am wavering between abdominal angina and mesenteric artery inadequacy. I think the latter is a possibility when we have demonstrated vascular disease, and he did have, I think, some vascular disease of the lower thoracic and upper abdominal aorta so that there is involvement in the area of the mesenteric artery.

The causes of the cerebral symptoms are very difficult for me to ascertain. There seem to be no localizing findings. We have evidence that the headache was not on the basis of increased intracranial pressure. Since this man was an alcoholic, I would like to consider the possibility of a subdural hematoma. I should think if it were the cause of this man's symptoms, there would be evidence of increased pressure.

DR. E. T. BELL: Could ischemia of the brain give him headaches?

DR. SMITH: Are you thinking of diffuse ischemia?

DR. BELL: Yes, I was thinking of a stenotic artery ischemia.

DR. SMITH: Yes, but he has had symptoms for some time, and I'm bothered by ascribing a severe headache to that cause. However, there is the possibility that he had an aneurysmal dilatation of some larger artery intracranially. That could cause headache without increased intracranial pressure. There is really no positive evidence for it and yet I feel that this man's headache has to be explained.

Now can we put all these things together in an Oslerian fashion? As has been done so often in this series, polyarteritis must be raised as a strong possibility. I agree that this is a possibility but I find no evidence for it. For me, it won't explain the headache very well. I think the hypertension is of long standing. It could explain the abdominal pain, but we already have two other reasons for vascular disease, and I dislike raising a third. Dissecting aneurysm should be considered. This could cause pain simulating myocardial infarction and it would give rise to diverse pains and findings elsewhere. I assume that the pulses were palpated and that they were equal. The man already has luetic aortitis. Others have reported that the finding of a dissecting aneurysm superimposed on the luetic aortitis is highly uncommon and actually the former gives some protection against dissection.

Embolization would be one way to put everything together—mycotic aneurysms from SBE or bland emboli from mural thrombus or from arteriosclerotic plaques. I should think, though, that we would have localized findings that are more specific.

I have to state that I am unable to arrive at a definite entity to explain his central nervous system findings, that I would favor an aneurysm of one of the large arteries at the base of the brain which was not seen roentgenographically.

DR. J. P. KNOEDLER: Do you think his headache could be due to hypertension?

DR. SMITH: His headache was of an acute onset, his hypertension chronic. I dislike ascribing his headache to hypertension.

DR. C. N. SADOFF: You said that you prefer to regard the hypertension as a chronic thing and would you then disregard the blood pressure reading in 1942?

DR. SMITH: No, I think that this hypertension probably developed in the interval.

DR. J. ROTHSTEIN: Would you entertain the diagnosis of a slow-leaking cerebral artery aneurysm?

DR. SMITH: I would be worried by the fact that he did not show cerebral spinal fluid changes.

QUESTION FROM AUDIENCE: Would you care to hazard a guess as to the most likely cause of his sudden demise?

DR. SMITH: Yes, I meant to mention it. His hemoglobin had fallen, for reasons not explained, when he came in. I think this amount would be too much for bleeding into the central nervous system. He died very suddenly. It sounds like a cardiac death or death from exsanguination; he is a candidate for either one.

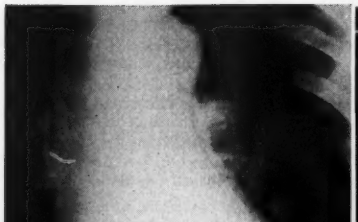

Fig. 1. Chest radiograph, April 16, 1951. Note widened ascending aorta with irregular linear streaks of calcium and second abnormal convex density superimposed behind enlarged left heart border. (Photograph retouched to give more accurate reproduction.)

Fig. 2. Heart, aorta and kidneys (one bisected). Severe atherosclerosis of aorta with aneurysmal dilation, ulcerations and mural thrombi. Coarse scars and pitting of kidneys.

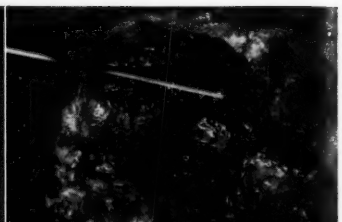

Fig. 3. Heart, aorta, closeup. Severe ulcerating atherosclerosis. Applicator stick in perforation which dissected into pericardial sac.

DR. NESBITT: Exsanguination from what?

DR. SMITH: It would be either from the aneurysm in the arch of the aorta or in the descending aorta.

DR. T. TAMLYN: Do you think that the cerebral difficulties might be explained by meningovascular lues?

DR. SMITH: Well, meningovascular lues should appear relatively early—within five years. If one requires absolute criteria for the diagnosis of vascular lues, he needs to demonstrate the organism. That would be unusual especially since he received some therapy.

QUESTION FROM AUDIENCE: About this radiologic sign of duodenal diverticulum; could that be considered as a perforation into fat rather than a true diverticulum?

DR. OLSON: It's on the lesser curvature of the duodenum. It almost has to be a diverticulum.

DR. NESBITT: Clinically, the diagnosis was that of luetic aneurysm. No one hazarded any suggestion on the chart as to the cause of the sudden exitus. The neurologist saw the patient on several occasions and all he offered as the cause of his headaches was "headache following spinal tap," which had been done several times in an effort to determine the cause of his headaches. Shall we read the students' diagnosis?

DR. E. B. FLINK: As a matter of fact, the students' opinions include those diagnoses that Dr. Smith has given. One just diagnosed atherosclerosis and arteriosclerotic heart disease. That's the briefest one. Most of them, though, include syphilitic aortitis with aneurysm. Many felt that the final episode was a cerebral vascular accident rather than cardiovascular.

Clinical Diagnosis

1. Leutic aneurysm.

Dr. Smith's Diagnoses

1. Leutic aortitis and aneurysm.
2. Hypertension, cause unknown.
3. Arteriosclerotic heart disease with coronary arteriosclerosis.
4. Cerebral arteriosclerosis; possible cerebral artery aneurysm.
5. Abdominal angina or mesenteric artery insufficiency.
6. Death from myocardial infarction or exsanguination from aneurysmal rupture.

Autopsy Conference
(October 22, 1954)

DR. D. F. GLEASON: The general autopsy examination revealed significant pathologic conditions only in the heart and aorta. The heart itself weighed 525 grams. The pericardial sac was tensely distended by approximately 750 cc. of blood. The left anterior descending coronary artery was completely occluded by an old, pale fibrotic and calcified process and there was a large area of thinning and scarring of the anterior wall of the left ventricle. The left circumflex coronary artery was approximately 50 per cent narrowed but the right coronary artery showed only minimal atherosclerosis. All of the cardiac valves were normal including the aortic valve. The striking pathologic lesion was in the aorta. The ascending and transverse portion of the arch of the aorta was dilated up to 8 cm. in diameter. This process began a few centimeters above the aortic valve ring. There was a very severe atherosclerosis involving the arch of the aorta and the entire thoracic and abdominal aorta and extending down into the iliac arteries as far as they could be examined. This was a very severe process with ulceration of the intima and patches of mural thrombus from the arch of the aorta to the iliac arteries. The aorta and heart are shown in the accompanying photograph.

From the gross examination alone, as seen in these pictures, we would not be able to make a diagnosis of syphilitic aortitis nor syphilitic aortic valvulitis. There is not a sharp line of demarcation of the process in the thoracic aorta as is usually seen with syphilitic aortitis. The aortic valves are entirely normal and do not show the thickened rolled valve edge nor the separation of the commissures usually found in syphilitic aortitis. However, the changes of atherosclerosis, if superimposed upon leutic aortitis, can obscure completely the changes we require to diagnose luetic aortitis.

The immediate cause of death can be seen in the photograph. There was a small rent in the wall of the

aorta some distance from the aortic valve but so located in the ascending aorta that the perforation extended into the pericardial sac. The pericardium contained almost 750 cc. of blood which was partially clotted. The patient undoubtedly suffered an acute cardiac tamponade due to rupture of the aorta into the pericardial sac.

The gall bladder and biliary ducts were normal.

Microscopic examination of sections of the aorta reveals a very severe degree of intimal atherosclerosis which extends deeply into the media and partially destroys it. In addition, there is a perivascular inflammatory infiltrate of lymphocytes and plasma cells around the vasa vasorum in the adventitia of the aorta and occasionally extending a short distance into the media. These are the lesions which are often described as characteristic of syphilis of the aorta but we know that severe atherosclerosis of the aorta can also be associated with such lesions, in the absence of syphilis. Sections of the myocardium confirm the presence of an old scarred infarct due to old occlusion of the left anterior descending coronary artery. In at least one area there are a few chronic inflammatory cells still present, consistent with the clinical duration of the infarct, that is three or four months before death.

Sections of the kidney reveal large irregular areas of atrophic kidney substance related to completely occluded small arteries. Many of these atheromatous lesions contain large "cholesterol clefts." We also see similar occluded arteries in the spleen and pancreas. The kidneys show only mild evidence of hypertension in the form of collagenous intimal thickening of small arteries and arterioles.

Sections of the lung reveal mild acute pulmonary edema and congestion.

Sections of the liver reveal no evidence of cirrhosis.

Gross and microscopic examination of the brain reveals a small area of relatively old encephalomalacia just adjacent to one of the caudate nuclei. This is in a stage of repair which would seem to indicate a duration of a month or two. This may have been partly responsible for the patient's headaches.

Final Anatomic Diagnoses

1. Severe generalized arteriosclerosis.
2. Severe atherosclerosis of the aorta from the ascending portion of the arch out into the iliac arteries.
3. Aneurysmal dilatation of the aorta.
4. Perforation of the ascending portion of the aorta into the pericardial sac.
5. Acute cardiac tamponade due to hemorrhage.
6. Luetic aortitis.
7. Diverticula of the duodenum and cecum and sigmoid colon.
8. Moderately severe arteriosclerotic damage to the kidney.
9. Relatively recent area of encephalomalacia in the brain.

We have to admit that we cannot be certain about the presence or absence of luetic aortitis, but considering all the evidence, we feel that it is probably present.

DR. SMITH: I would like to ask whether the ostium of the superior mesenteric artery was occluded by atherosclerosis.

DR. GLEASON: I am sorry, but that area was not described specifically and I cannot identify it from our gross photographs.

DR. BELL: I would like to comment upon this aneurysm. When syphilitic aneurysm is overshadowed by massive atherosclerosis like this, we do not know whether syphilis is present or not. We have no criteria to identify it when severe atherosclerosis is present. If you read that some writer diagnoses syphilis or no syphilis in cases like this, I tend not to believe him. I do not believe there is any way to tell them apart. If syphilitic aortitis occurs in a patient without much atherosclerosis, we can make the diagnosis relatively easily. This man has an extremely severe atherosclerosis which has destroyed much of his aorta. That can cause dilation also, but in view of this aneurysm being in the ascending arch and in the presence of positive serology, I believe that the presumption is that this has a syphilitic background to it. Any kind of an aneurysm can rupture.

DR. SMITH: I would like to ask about pain in small cerebral infarcts. I have the impression that the headaches associated with these are not unremitting nor unrelieved by codeine. That bothers me a great deal. I know we do not have an explanation for the headache otherwise. I wish we did.

DR. BELL: The patient probably had a lot of cerebral ischemia. The headache might be due to that.

DR. SMITH: Other individuals with cerebral ischemia do not usually have headache.

DR. FLINK: Would you say just a word about the causes of sudden death?

DR. SMITH: When sudden death occurs, that is within a few minutes, it is almost always cardiovascular. Death due to lesions of the central nervous system are very infrequently that fast. This patient was observed about half an hour before the comatose state and he died very promptly after that. There are only a few central lesions which will cause sudden death, one being rupture of an aneurysm of the basilar arteries; it is uncommon.

DR. FLINK: Incidentally, one of the students did give rupture of an aneurysm of a basilar artery as the cause of death. We are often faced, in patients of this age, with the problem of explaining an elevated sedimentation rate, in a patient who is otherwise apparently well. Seeing the aorta in this man, I just wonder whether there is not enough thrombotic process and enough ulceration to explain the elevated sedimentation rate. I am sure he had myocardial infarction in the not-too-distant past and he has destruction of tissue in the kidney, but I wonder whether the aorta does not account for the elevation of sedimentation rate in this case.

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President's Letter

TO GIVE WISELY

The problem of personal giving to all the many and varied appeals for funds that come, not only to the doctor's office, but to the home of the average citizen of our state, is one that is becoming increasingly more acute. In scores of emotional appeals, many coming by mail from obscure institutions and faraway places, we are besieged to contribute to every conceivable area of good will and human need.

Americans have a reputation for generous giving, for caring for their fellow men, but this also makes us vulnerable targets for fund campaigns, some of whose natures, purposes, and methods, are questionable. We find it virtually impossible to give to all of the worthy drives.

Because organized fund raising has become big business—money-wise it is the fourth largest industry in the nation—the average citizen wonders if he can depend on his judgment alone to determine the campaign merits. He knows that most campaigns are masterminded by skilled professionals, hired for their scientific know-how about how to open the purse strings. For better or worse, these professionals are a necessity in this modern age, and we all realize this. We know that the future of great humanitarian endeavors hinges on how successfully a campaign is put across.

There is a great overlapping of appeals for funds by various organizations. This is particularly true in the medical field, where societies have been formed to establish research funds and to relieve sufferers in almost every conceivable disease. The comedian, George Gobel, recently remarked that it is getting now so that there are hardly any good diseases left. Would it be possible to consolidate all the money raised for research into one fund?

The disturbing problem is pointed out by the following finding in a report from the Campaign Review Council in the city of Duluth. There are thirty-four separate groups now seeking public support in Duluth to aid crippled children and adults. Some of these groups are covered by the Community Chest; most of the others conduct private fund-raising campaigns. Some of the thirty-four groups are concerned only with research; others with rehabilitation; still others with giving personal aid. All are worthy causes, but where does the average citizen draw the line?

As doctors, we are often asked whether the funds raised by the various organizations are well supervised and well spent. I'm sure that we can honestly say that in the great majority of instances, the money received is well spent. However, the very multiplicity of all these campaigns will eventually lead to a point of diminishing returns.

Wouldn't it be possible for each of us in our own communities to work toward some form of consolidation of these various fund drives? In Duluth, for example there is one large fund drive occurring in almost every month of the year, not to mention many of the rather questionable requests for funds that are received from organizations outside our state at or near the holiday season. It is my fear that the very multiplicity of these drives will be the factor that "kills the goose that lays the golden egg."

Arnold O. Swenson

President, Minnesota State Medical Association

Editorial

JOHN F. BRIGGS, M.D.
HENRY G. MOEHRING, M.D.
ARTHUR H. WELLS, M.D.

TAKING NOTES AT MEDICAL MEETINGS

We have been impressed recently, not only with the tremendous number of note-taking techniques employed at medical meetings, but with the startling revolution our own note-taking has undergone. There was the day when we went to medical meetings with much note-taking paraphernalia including at least two never-fail mechanical pencils, that always managed to fail, and against the failure of which we also carried a freshly filled fountain pen. (In reviewing some of our old notes, however, it appears that a pencil can fail and the writer not become aware of it, for some of our notes appear only as indentations in the paper—no lead, notes taken in the dark.)

Then there was a refinement in note-taking; the flashlight. This required that a flashlight be added to a pocket already bulging with writing gear. When the lecturer demanded that the lights be turned out so he could show his slide, the note-taker grasped the flashlight and its switch in the left hand, his pencil (we assume it is still functioning) in the right hand, and somewhere between elbows, left forearm, knees and anterior aspects of thighs, managed to grip the note-book —and proceeded to take notes. There was always something satisfying in writing prettily under these circumstances; many of our notes were prettier than what we do sitting à la Palmer method at a desk. But a super-refinement came. The combination pencil (or pen) and flashlight. This took the whole note-taking business out of the realms of technique and reduced it to a semi-automatic mechanical process. It left too much time to think about the subject matter and has led directly to the degeneration of our note-taking. Degenerate it did from here on.

As soon as our mind was occupied only with what the lecturer was saying and doing, and the mechanics of note-taking no longer provoked the invigorating thrill of masterly exercise of a technical skill, we found our notes going along well for a line or so, then the words became more scrawly; finally, the words trailed to the vanishing point and were nothing, absolutely nothing. This was not as unsettling as it sounds, for we soon developed a lethargic, contented sort of satisfaction in the technique of dozing in a well-illuminated room without the lecturer or neighbors giving overt evidence of being aware of our sleeping. True, once in a while our technique slipped, and a neighbor was constrained to nudge an interruption to what he would later allege was noisy respiration, but, in a general way, we continued to bask in the self-satisfaction that comes from attending lots of medical meetings, *der ewige Student* type of reputation. Note-taking in itself, you see, had become as effortless as we'd like to have everything else in this life.

Disquieting news reaches our ears now, though. We've been told about people who go to meetings, take notes, rush to their hotel rooms, and transcribe the notes immediately! Now these folks are going to get ahead of us. They will have not only the reputation of going to great numbers of medical meetings, but will be insufferable enough to bring around their well-written notes, and, what's worse, be able to interpret them. This blow has been softened by the underground intelligence that slow decay is tanglefooting the adherents of this school. They have taken to transcribing their notes to the accompaniment of a long, cool drink —after a hot, tiring day in the lecture hall, you know—and the net result is reported to be like that in the system we have; i.e., first line or two is wonderfully written and then the words gradually become as blurred as the thoughts they express.

The other source of disquiet is in line with the reports of the colleagues who take portable dictating machines in their automobiles and dictate patients' histories while en route to or from house calls, weekends, and such like; or the folks who have machines to read tape-recorded medical literature to them while they drive. Insofar as note-taking goes, this type of efficient perfectionist rents a dictating machine at the meeting place, takes notes, rushes to hotel room, transcribes

notes onto recording disc or belt, mails record home to his secretary, and has the TYPED notes waiting on his desk when he gets back. This is nothing if it isn't unsporting and, though we are terribly, terribly hurt by this win-regardless-of-cost attitude, we shall simply ignore this school of note-taking.

This brings us back to the good old method requiring no special equipment, available to all of us from the lowest type of specialist to the highest type of provincial physician, namely, alternate dozing and note-taking with a blunt stub of a pencil on the back of a tattered envelope. It gives you just as much satisfaction as any other method, takes just as much know-how, and will be read and referred to just as often and with equal yield as any flossier technique. And what's more, it gives time to observe that alert gent in the front row who listens eagerly, whose eyes fairly glitter with the joy of the game, and who gets up after the talk and, with obvious enjoyment, intelligently punches holes in the lecture and the lecturer.

H.G.M.

THE VALUE OF SPECIALTY BOARDS

With the unprecedented growth of medical science in the twentieth century came the pressing need for specialization to encompass newer and more refined developments in practice. Traditionally, Vienna and Berlin became the mecca for hordes of American physicians seeking postgraduate training. While many of these men were soundly grounded and motivated, the lack of standards and of measured attainment led to a sense of insecurity in the total program. Too frequently the sole evidence of special training was a series of patently meretricious certificates in the physician's office.

American medicine became increasingly aware of the necessity of keeping its house clean. As in other respects of public welfare, recourse to moral suasion rather than legal action was sought. The movement for the regularization of specialty training was initiated in 1916 by the foundation of the American Board for Ophthalmic Examination. Its terms of limitation were expressed as follows: "Each certificate granted or issued does not of itself confer or purport to confer upon any person any degree or legal qualifications, privileges or license to practice ophthalmology. Nor does the Board intend in any way to interfere with or limit the professional activities of any duly licensed physician." In the intervening years, eighteen additional specialty boards have been established, and, as in the case of the American Board of Ophthalmology (revised designation), recurrent expressions of the designs in the certification of specialists have been made. For example, the American Board of Surgery stated, "The prime object of the Board is to pass judgment on the education and training of broadly competent and responsible surgeons, not who shall or shall not perform surgical operations. The Board especially disclaims interest in or recognition of differential emoluments that may be based upon certification."

The Advisory Board for Medical Specialties, established in 1934, has co-operated with the Council on Medical Education and Hospitals of the American Medical Association in guiding the foundation of new boards and in establishing broad, general principles of procedure. However, the individual requirements of the several Boards have been derived without external influence. For example, in the early operation of the American Board of Internal Medicine, the conventional formula for admission to the examination was three years of residency and two years of the practice of internal medicine after graduation from a Class A medical school and one year of internship. After a period of years, the experience of the Board dictated a much more liberal policy, so that currently it is possible for a candidate to be admitted to this examination under one of several plans of preparation. Indeed, twelve years of the practice of internal medicine after the internship without a formal residency is considered a sufficient discipline. The ability of the candidate rather than the strictures of arbitrary terms of sharply defined service establishes his eligibility for certification.

By common consent, the specialty boards have contributed immeasurably to the improvement of the quality of medical service. The standards of specialty training in this country have improved apace as was anticipated when the several Boards were established. In a period when continental medicine deteriorated to an all-time low, the curve of medical education in this country showed an up-swing. In a material degree, this advance can

be directly or indirectly traced to the movement for certification in the specialties. With the available facilities, new incentives for professional advancement were afforded a greater number of physicians. Furthermore, direction was lent to graduate training where none had existed before the advent of this program.

An overlooked dividend of certification in the medical specialties is the requirement of basic qualifications for candidates in the sub-specialties. In an earlier day, the acquisition of a gastroscope and a short course in gastroscopy abroad sufficed to qualify a physician in gastroenterology. The purchase of an electrocardiographic apparatus was tantamount to recognition as a cardiologist. Now such candidates must prove their adequacy in internal medicine before they can be admitted to the examination in the medical sub-specialty. By this simple expedient, the standards in the medical sub-specialties have been immeasurably advanced. Never has the American public been so well protected in the insurance of the qualifications of the practitioners in the various specialties of medicine and surgery.

Unfortunately, certification in medicine and surgery has not been an unalloyed blessing. Perhaps its greatest drawback has been the curious psychologic reaction of "working for the Boards." Perchance this response of immature minds is inevitable. Specialty boards set minimal standards of professional adequacy. Any interference with the continuing education and sustained growth of physicians must be met by positive measures. Unfortunately, the yardstick of certification is applied to the appointment and advancement of physicians in various capacities. The most pernicious device in its utilization has been its requirement by certain hospitals for staff appointment in spite of the expressed design of the several specialty boards. Conversely, the lack of certification has been used as a lever to deny staff appointments or to remove proved clinicians from established posts. Such abuses of the medium of certification are as unfair as they are reprehensible. Perhaps the leveling influence of time and experience is required in all good works.

The principle of supply and demand will unquestionably dictate changes in the programs of specialty training. Certification is a means to the end and not the end itself. The mechanism for rating the qualifications of specialists will vary widely among the several branches of medicine and surgery. Any formula that improves the quality of medical service should be encouraged; but the worthy efforts to broaden the base of training and to encourage the continued growth of specialists must not be submerged by the specious and hollow objective of mere certification.

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CHILD PSYCHIATRY

The rapidly growing field of social psychiatry has developed largely from the application of psychoanalytic theory to general behavior. This has changed considerably the nature of psychiatric practice. The psychiatrist, with few exceptions, no longer limits his work to the treatment of the more serious disturbances such as psychosis and psychoneurosis. He plays a more active rôle in the treatment of the many social and emotional maladjustments to life, and these include the broad fields of marital adjustment, delinquency, psychosomatic illness, and the art of living.

Research in the field of human behavior has demonstrated that the way a person feels, believes and behaves in adult life is largely conditioned by the early emotional experiences to which he was subjected. This has placed tremendous importance on the early years of life and has provided a large body of facts concerning the early development of personality.

There has been considerable research recently on the first few months of the infant's life, growing out of the observation that emotional pathology in the young child is due to the absence of a strong positive emotional tie between the infant and his mother. We are beginning to understand early childhood psychosis much better since this research began and we are rapidly developing a treatment program to deal with this condition. The facts gathered from this research have already been extended to other emotional problems of early childhood which are becoming more clearly understood for the first time.

Studies of children, as revealed through child guidance clinic work, demonstrate that children of all ages develop emotional pathologic conditions when there is something wrong in the relationship between them and their parents.

Rarely does one see a child who presents emotional pathologic conditions of any nature—whether it be night terrors, soiling, school failure, inability to get along with other children, hostile aggression, or delinquency—where the emotional relationship between the child and his parents is a healthy one. Almost invariably there is conflict between the parents, growing out of incompatibility, disloyalty, or the many other forms of family pathologic situations.

As a result, in order to effectively treat the emotional conflict or illness of the child, one must work intensively with the parent-child relationship, the problems in the parents themselves, and any problems created within the family. The best way to deal effectively with these problems is for the so-called team of social worker, clinical psychologist and psychiatrist to work together intensively with the child and his parents and all the many emotionally charged situations to which the child is subjected. This task can seldom be accomplished by the child psychiatrist alone; it is too complex and requires the skills of the several disciplines.

Recently, group therapy has been added to the psychiatric treatment of the child and we have found this to be an invaluable aid in the understanding of a child as he relates himself to other children.

The body of information required to carry on psychotherapy of children intelligently is so extensive, and the particular skills in understanding and helping a child are so important that a long period of intensive training is absolutely essential before a psychiatrist is prepared to do psychiatric treatment work with children.

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ANNUAL MEETING

The 102nd annual meeting of the Minnesota State Medical Association will be held in Minneapolis, May 23-25, at the time of the 100th anniversary of the Hennepin County Medical Society.

Business sessions of the Council and House of Delegates will precede the scientific sessions which will begin Monday with meetings in the Minneapolis Auditorium. A tentative program will appear in the April number of MINNESOTA MEDICINE, and the official program should reach members about May 1.

THE VALUE OF A SAFETY DEPOSIT BOX

Century after century, down through the ages, the primary concern of man, after providing a livelihood for himself and his family, has been the protection of the surplus wealth accumulated as a result of his labor. Closely paralleling the development of the family unit into an organized society, the protection of the concentrated money of the community has been the responsibility of the banker. With this responsibility arose the problem of creating a depository which would safeguard the money and valuables entrusted to his care.

Ingenious, yet crude were the early methods employed, but from these humble beginnings evolved the modern steel vault, equipped with protective devices and guarded by massive doors weighing many tons, which today make the vault a marvel of safety and convenience, storm-proof, burglar-proof and fire-proof—safe against almost any act of nature or man.

The modern bank, by popular demand, has incorporated in its security function a deposit section containing thousands of safe deposit boxes in varying sizes which are available to the general public. It is not difficult to conceive the value of a safe deposit box for private use when it is realized that the legal evidence of much that mankind possesses, is on paper—paper which if lost, stolen or destroyed, might be impossible to replace. Insurance will help rebuild a home, purchase a new car, furniture or clothing after a catastrophe, but there are many items of intrinsic or sentimental value together with irreplaceable papers and documents which money can never restore.

The use of the safe deposit box is limited only by the imagination of the holder; it is not only a convenience but a means of protection also frequently utilized as an important tool in managing financial affairs in a systematic manner. It is peace-of-mind insurance for the individual, partnership or corporation—one of the most important yet comparatively inexpensive banking services offered.

ROLLIN O. BISHOP
President, American National Bank
Saint Paul, Minnesota

The Dean's Page

UNDERGRADUATE EDUCATION FOR GENERAL PRACTICE

The trend toward specialization in medicine has been discussed with increasing frequency in recent years, and more often than not it has been deplored. Yet medicine's growing complexity suggests that this trend will not be reversed. On the other hand, the well-trained general practitioner will continue to play a vital role in the care of the sick. There can never be an acceptable substitute for the close personal relationship between a patient and his "family doctor."

The principal objective of a medical school is to provide its students with the basic tools essential for general practice. The same tools are equally necessary for the future specialist, teacher, or investigator. To the end of providing their students with a better background for medical practice, a number of schools have recently introduced changes in their curricula, for example, "integrated" teaching, family care plans, preceptorships. Although differing in their forms, each of these programs represents an attempt to give the student a broad view of medicine rather than a series of compartmentalized peeks.

Tradition and the appreciation of socio-economic realities allow us but four years for the formal education of a physician—a period unchanged in more than four decades. Yet today's student must assimilate an infinitely greater amount of information than did the student of 1915, considerably greater even than did the student of 1940. Two closely related conclusions may be drawn from this. First, medical students must be made aware that their education is, in fact, a life-long process and that their years in medical school represent only its beginning; and second, medical schools must plan their curricula with this in mind.

In light of an ever-expanding body of medical knowledge and a fixed period for formal undergraduate training, how may a medical school make best use of the four years during which its faculty is responsible for the education of its students? In our opinion, it must see that before graduation its students acquire the following:

1. A comprehensive knowledge of the basic medical sciences. If a student fails to acquire a real understanding of the basic sciences in medical school, it is most unlikely that he will do so after graduation.

2. The fundamentals of clinical practice in each of the various fields. This means that the student must be taught primarily the approach that is required in solving clinical problems.

3. An appreciation of the role of research in the continuing growth of medicine.

4. A knowledge of how to use medical literature effectively and, along with this, the background and attitude necessary for the critical evaluation of reports of new fundamental, diagnostic, and therapeutic developments.

5. An appreciation of the absolute necessity for integrity and adherence to the ethical code in the practice of medicine and a realization that the physician must accept civic and social responsibilities apart from his professional responsibilities.

6. An awareness of the various paramedical agencies, for example, public health, social service, occupational therapy, which can help him in rendering maximum service to his patients.

If we accept the foregoing as reasonable objectives for the medical school, two corollaries follows: (1) The general caliber of the internship, as an integral part of medical education, must be improved; and (2) The practicing physician will have to make increasing use of the various forms of continuing medical education.

In summary, the best undergraduate teaching program is one which will furnish the student a sound foundation on which he may base his future training. His personal preferences and interests may take him into general practice, specialty practice, teaching and research, public health, or administration. Regardless of his later choice, such a background will make him a better physician.

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Medical Economics

Edited by the
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HEALTH MESSAGE SHOWS LITTLE CHANGE

President Eisenhower's health message to the Congress carried no recommendations that had not been made earlier in his State of the Union or budget messages, except his request for a bigger U. S. contribution to World Health Organization. The AMA congratulated the President on his statement that health proposals "recognize the primacy of local and state responsibility" and would "encourage private efforts with private funds."

His comments and basic outlines for health programs are:

Reinsurance.—The Administration wants Congress to appropriate $25 million to start a federal reinsurance fund for the use of voluntary health insurance groups. The President said reinsurance would be most helpful in three broad areas (a) with rural families, (b) to expand catastrophic insurance, and (c) to bring protection to low-income families in the form of coverage for home and office calls as well as hospitalization. Last year the House defeated a reinsurance bill that followed the same lines but that did not pinpoint certain areas. This was introduced in the omnibus health bill S. 886.

The AMA's official comment on this proposal was that the AMA has been in complete accord with the stated purpose of legislation designed to promote voluntary health insurance and commended the President for his beliefs and efforts in encouraging its expansion. However, the AMA still believes that the proposed reinsurance system will not achieve the desired results.

Medical Care for Indigents.—The President described the present medical care program for public assistance recipients as "far from adequate." He proposed $20 million more in U. S. appropriations, to be matched by the states and placed in a separate fund exclusively for medical care payments. This fiscal year the U. S. is spending about $80 million in this field. The AMA was not prepared to comment on this proposal yet.

Mortgage Guarantee.—The Administration is offering an amended version of last year's Kaiser-Wolverton bill for U. S. guarantee of private loans for health facilities. The President said: "Present methods of financing are not always satisfactory in meeting this problem. Many sponsors and operators are unable to qualify for grants under the recently extended hospital survey and construction act (Hill-Burton). Sponsors of health facilities often find it difficult to obtain private capital for construction." The AMA said it has no opinion yet on this measure.

Health Personnel.—Three methods are proposed to increase the supply of trained personnel, (a) grants to states for training practical nurses, (b) Public Health traineeships for graduate nurses in specialty nursing fields, and (c) establishment of PHS traineeships in public health specialties, including mental health. The AMA commended the President for his support of these proposals.

Mental Health.—Three approaches recommended (a) more aid to states and communities, (b) more money to train personnel for care of mental patients under present authorizations, (c) a new program of project grants aimed at improving the quality of care in institutions and searching out ways of reducing length of stays. The latter point is in recognition of the fact that the care of mental patients is the heaviest financial burden in the medical fields for most states. The AMA agreed with the President on this matter also.

Also included in the President's recommendations to Congress is the extension of the "Doctor-Draft" act. This is incorporated in H.R. 2886, which simply extends the act for two years.

PRESIDENT'S BUDGET IS EXTENSIVE, EXPENSIVE

The Administration has presented Congress its formal request for funds to maintain all programs, including health and medical, for the 1956 fiscal year starting July 1, 1955. The total request for the Department of Health, Education and Welfare is $2 billion, just a slight increase over the current year's estimated spending.

Included in this amount is the request for Hill-Burton hospital construction funds amounting to $125 million. This will be for new facilities and grants to complete hospitals now under construction.

Increased funds were also requested for research projects, some as much as 50 per cent.

VA Medical Care Costs

Medical care costs for the Veterans Administration were set at almost $889 million, compared with $862 million in current spending; hospital construction costs were set at $60 million (mostly remodeling) as compared to the current $50 million.

In his budget message the President expressed concern that the VA patient load would rise 4 per cent next year and that:

". . . more than two-thirds of the expenditures will still be for patients hospitalized or treated for ailments not connected with military service. . . . we must bear in mind that government policies designed to assist in the maintenance of a prosperous economy and to support social security, health, and other humanitarian programs are all of value to veterans as well as to other people. Since more than two-fifths of all adult males are entitled to veterans' benefits, expenditures for veterans are a budgetary problem of major interest to the whole population."

STANDARD HEALTH INSURANCE PLAN PROPOSED FOR U.S. WORKERS

The Administration has revised its plan for health insurance for federal employes and is proposing a three-way option for them: participating in (1) a new standard, nationwide plan, (2) existing plans sponsored by their own national employe organizations, or (3) local plans offered in their community or through local employe unions or other agencies.

The standard plan would offer catastrophic or major medical care insurance. It provides:

1. U. S. estimated contribution would be $55 million a year, paying up to one-third of premium costs, with a limit of $1.50 per month for employes without dependents and $4 per month for those with dependents. If the employe chooses the standard national coverage, the cost to him would be $3 per month if he has no dependents and $8 per month if he has dependents. On other types of coverage, the employe's payments would vary with the premiums, but U. S. contribution would still not exceed the above figures.

2. The standard plan would be straight cash indemnity based on a schedule of benefits set by the Civil Service Commission.

3. Major medical cost protection would be restricted. After the policyholder had exhausted the benefits of the basic policy, he would have to meet the next $100 of expenses himself. Then, the standard policy would reimburse him for 75 per cent of the costs up to a maximum of $2,500.

4. After a minimum of fifteen years' civilian service, employes who retire would be permitted to continue under the program by authorizing premium deductions from their retirement checks.

5. Civil Service Commission would be responsible for administration of the entire program, but would contract with a single insurance company to operate the national standard plan. It would not become operable, however, unless 600,000 of the 1,800,000 eligible government workers agree to participate.

COMPULSORY HEALTH INSURANCE AGAIN INTRODUCED

H.R. 95 is a verbatum copy of H. R. 1817 introduced in the 83rd Congress two years ago, providing, among other things, for national compulsory prepaid personal health insurance. Its author is Rep. Dingell of Michigan, author of the previous bill also. It also provides for subsidies for federal aid to medical education, medical research, hospital construction act amendments, special aid for rural and other shortage areas, state grants for local public health units, research in child life and additional grants for maternal and child health and crippled children's services.

M.D. SHORTAGE IS A FADING SPECTER

A recent editorial in *Medical Economics,* the monthly magazine, notes the rapid rise in the number of medical schools in the nation, to help refute the idea that there is an acute doctor shortage in America.

The editorial stated: "For years there's been agitated debate about a national doctor shortage. Medical men couldn't stop it by pointing to the increasing size of graduating classes pouring forth from existing medical schools."

Reasons given were that the population was increasing just as fast. For, between 1940 and 1950 the total number of physicians in this country increased by 15 per cent; but the total population increased 15 per cent too.

But, the editorial notes what is happening today: Two new medical schools (the University of North Carolina and the University of Puerto Rico) produced their first graduating classes within the past year. Eight more new schools (U.C.L.A., Albert Einstein, Seton Hall, and the universities of Miami, Missouri, Mississippi, West Virginia, and Florida) are ready for early training of doctors.

The editorial quotes statisticians as believing then "the rise in the number of medical graduates should be relatively more rapid than general population increases in the course of the next six to ten years."

In conclusion, the *Medical Economics* editorial says:

"This heartening break-through doesn't solve *all* our doctor-supply problems. There's an unquestioned shortage in certain local areas and in certain key specialties. But at least the specter of a ***national*** doctor shortage should begin to fade."

BUSINESS HAS AN INTEREST IN HEALTH

According to Benson Ford, president of the Board of Trustees of Detroit's 650-bed Henry Ford Hospital, business has a substantial interest in health and health problems throughout America. Mr. Ford is also vice president of the Lincoln-Mercury division of Ford Motor company.

In a speech at the 56th annual convention of the American Hospital Association, quoted in the December, 1954 issue of *Hospitals,* Mr. Ford said that "of the 43 million Americans who take part in Blue Cross, a large majority are covered by industrial group plans. Industry also foots part of the bill for millions more Americans in other insurance plans. Add to that the cost of private industrial medicine, insurance against industrial accidents and illness, and it becomes clear that business has a substantial interest in the whole health system of our country.

Mr. Ford outlined the broader problem of union demands on industry, the aging population and other problems which affect industry's interest in health matters. But he stated:

"Up to this point, I seem to have painted a picture of a pretty sadly-confused health situation. That would hardly be a true or fair picture. Most of our present difficulties are inherent in the nature of the private enterprise system. Ironically, it is the very high quality of our private medicine and our partial success in developing private means of financing medical care that make it possible for us to enjoy some of these worries I've been talking about."

Suggests Permanent Health Commission

Mr. Ford suggested a solution to the national problems of health matters in a health commission representing all the major segments of the health profession, to examine our major health needs and to recommend how best these needs can be met.

"I'm not suggesting a super-lobby to fight socialized medicine or a 'politburo' to dictate hospital and medical practices," Mr. Ford continued. "I have in mind a top-level professional group adequately staffed to study the developing pattern of wants and demands and to establish standards and practices for the guidance of all our services, including hospitals, clinics, medical schools and medical research centers."

In conclusion, he said, "I think we must all recognize the existence of a broad public desire for a kind of health care commensurate with the vast wealth and resources of our nation." He declared:

"If you, the professionals of health, can supply the drive and leadership to get that job under way, I am sure you will win the earnest gratitude and also the eager co-operation of industry, of labor, of government and of all the American people in helping you solve a great public problem by private action."

Public Health

DUTIES OF THE PHYSICIAN IN REGISTERING BIRTHS AND DEATHS

The physician is the first person to take official note of a birth or a death. On him, therefore, rests the responsibility for prompt and accurate reporting of these events occurring in his practice. Laws regarding the reporting of vital events are changed from time to time. For the help and guidance of physicians, the Minnesota State Board of Health herewith presents the current requirements.

The Vital Statistics law requires the physician to:

(a) File a birth certificate for every live birth he attends.

(b) Certify the cause of death when he is the physician last in attendance, unless the case is under coroner's jurisdiction.

Birth Certificates.—The Vital Statistics law requires the attending physician to file, within five days, with the local registrar of the district where birth occurs, a birth certificate for every live-born infant he has attended.

Exception.—A birth certificate for a child *born out of wedlock must be filed directly and promptly with the state registrar,* 469 State Office Building, St. Paul 1, Minnesota. (Local registrars are clerks of the District Court, except in cities which by ordinance maintain a primary registration district. In such cities, of which there are twenty-two in Minnesota at present, the health officer is the local registrar.)

Reportable Births.—Live births and stillbirths are reportable after twenty weeks' gestation.

Live Births.—The standard definition of a live birth is "an infant which either breathes or demonstrates heart beat or movement of voluntary muscle after it is completely outside the body of the mother." Live births are to be reported on the *Live Birth* certificate; stillbirths on the *Stillbirth* certificate.

Hospital Births.—A birth record is a legal record and is of great importance to the person throughout his life. When hospital personnel prepare birth records for the physician, he is nevertheless responsible for their legibility, completeness, and verity. He must sign them in his own handwriting. They must also be dated. If the physician is not satisfied with their appearance or content, he should require that they be made out again before he signs them.

Stillbirths.—If a funeral director takes charge of the body of a stillborn infant, the law requires him to prepare a certificate and to present it to the physician for certification of the causes indicated. If there is no funeral director, the hospital or the physician may take the initiative in preparation of the certificate.

Death Certificates.—The funeral director is required to present the death certificate to the physician last in attendance, for certification of the cause of death, unless the coroner has jurisdiction, in which event the certificate must be presented to the coroner. Physicians and coroners should promptly certify the cause of death and immediately thereafter return the certificate to the funeral director so that he can file it and obtain the required permits before burial or other disposition of the body.

Cause of Death.—Cause of death should be certified in full (abbreviations should not be used) according to directions set forth in the World Health Bulletin, Medical Certification of Cause of Death, obtainable from the Section of Vital Statistics, Minnesota Department of Health, 469 State Office Building, St. Paul 1.

Autopsy Pending or Diagnosis Deferred.—If autopsy is pending or diagnosis is deferred for some other reason, the physician or coroner should so state in the death certificate. He should date and sign the certificate and return it at once to the funeral director. At a later date, the state registrar will obtain a statement of the cause of death from the physician or the coroner.

It may be of interest to physicians to know that Minnesota rated 99.9 per cent completeness in the registration of births in the 1950 birth registration test conducted by the Bureau of the Census in co-operation with the National Office of Vital Statistics and the Vital Registration Executives of the United States. Minnesota was tied with Rhode Island for second place among the states, exceeded only by Connecticut with a rating of 100 per cent. Credit for this achievement was due to the physicians, hospital personnel, and local registrars of Minnesota who have most faithfully fulfilled their legal obligations regarding birth registrations. During the test period studied, only two Minnesota physicians failed to report. Constant co-operation between local and state officials is necessary if the high standard of vital reporting in this state is to be maintained.

J. W. Brower
Director, Division of Departmental Administration
Minnesota Department of Health

Current Concepts

DIET AND ATHEROSCLEROSIS

ANCEL KEYS, Ph.D.
Minneapolis, Minnesota

The theory that the diet may affect the development of coronary heart disease is not new. For years, the balance between pro and con teetered under the accumulation of indecisive evidence from animal experiments, often having little relevance to the real problem in man. Recently, however, the increasing dominance of coronary heart disease in our national health picture has stimulated much more research, and some of this has been more systematic and more directly concentrated on man himself. The result is that we can now see some of the outlines, if not the details, of a major relationship between the habitual diet and the tendency towards atherosclerosis and resultant coronary heart disease.

Perhaps the most crucial evidence is that human populations who subsist on different diets show great differences in the prevalence of clinical coronary heart disease, in the age-specific mortality ascribed to coronary or degenerative heart disease, and in the incidence of atherosclerosis seen at autopsy. The old impression that coronary heart disease may be rare in some populations, such as the Chinese, has been replaced with conclusive data. It is now clear, for example, that middle-aged men in the United States have perhaps ten times as much atherosclerosis, and die far more frequently from coronary heart disease, than do South African Bantu, or Japanese of the same age. It is not simply that white men are more afflicted than men of the Mongol or Negroid races, because there are also notable differences between white men. We may contrast Minnesotans with Italians or Spaniards, for example, and point to differences between white men in the same country, such as rich versus poor Spaniards, or Italians in Naples versus Italians in Bologna. It is most significant that the indicated differences between populations in regard to coronary heart disease appear to be parallel to differences in the average concentration of cholesterol in the serum in samples of these populations.

The unifying concept that brings order into an otherwise mystifying picture is that the proportion of fat in the diet has a powerful effect on the serum cholesterol level and so on the development of atherosclerosis. The dietary cholesterol itself is unimportant, but whether different neutral fats in the diet have different effects is as yet undecided. The important point is that all of the data —and they come from Guatemala, Sweden, and England as well as from Spain, Italy, South Africa, Japan and the United States—show a remarkably regular and direct relationship between the total fat content of the diet and the prevalence of coronary heart disease at given age. Further, the experiences of World War II and after, when many countries changed their diets, are in close agreement and indicate that dietary changes may have large effects on mortality in populations in a year or so.

It is significant, then, to note that our average American diet is the world's highest in fat and that the proportion of our dietary calories supplied from fats has been steadily increasing for a long time. Contrast our present more than 40 per cent of calories from fats with the 20 per cent figure for the Neopolitan or the 8 per cent figure for the Japanese!

As yet, there is little evidence that dietary manipulation will greatly change the prognosis of the coronary patient, though time may change this picture, but it is already reasonable to hope for real progress towards prevention through dietary means. Obviously, we need much more research, particularly systematic studies on populations which can tell us things not to be learned from our laboratory animals, but the future ahead looks bright. We can now be sure that age alone does not inevitably bring atherosclerosis and coronary heart disease.

It is clearly evident that our public health problems of the future will be concentrated among older persons, among whom chronic disease and disability are more prevalent; their social and economic overtones will have an impact on almost every family. Our plans must encompass the degenerative diseases, the long-term illnesses, and the disabling conditions of older age groups. This does not mean that we can relax our guard against communicable diseases, nor lessen our campaign to reduce infant and maternal mortality, nor let up on our crusade to eradicate tuberculosis. Environmental sanitation is as important as ever and requires constant vigilance to keep the air, water, food, and milk free from pollutants including those spawned by atomic fission. HERMAN E. HILLEBOE, M.D., *New York State Journal of Medicine*, December, 1954.

Reports and Announcements

MEDICAL MEETINGS

State

MINNESOTA STATE MEDICAL ASSOCIATION, annual meeting, Minneapolis, May 23-25, 1955.

National

American Association of Blood Banks, eighth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

American Cancer Society, Board of Directors and special committee meetings, Hotel Radisson, Minneapolis, June 2, 1955.

American College of Gastroenterology, southern regional meeting, Hotel Peabody, Memphis, Tennessee, April 24, 1955.

American College of Surgeons, sectional meeting, The Fort Harry, Winnipeg, Manitoba, Canada, April 25-26, 1955.

American Goiter Association, annual meeting Skirvin Hotel, Oklahoma City, Oklahoma, April 28-30, 1955.

American Medical Association, annual meeting, Atlantic City, New Jersey, June 6-10, 1955.

American Radium Society, Hotel Shoreham, Washington, D. C., April 21-23, 1955.

Gill Memorial Eye, Ear and Throat Hospital, twenty-eighth annual spring congress, Roanoke, Virginia, April 4-9, 1955.

Industrial Health Conference, Memorial Auditorium, Buffalo, New York, April 23-29, 1955.

Society for the Prevention of Asphyxial Death, New York Academy of Sciences, New York, New York, March 24, 1955.

International

Inter-American Congress of Radiology, Shoreham Hotel, Washington, D. C., April 24-29, 1955.

International Hospital Congress, Lucerne, Switzerland, May 30-June 3, 1955.

INTERNATIONAL COLLEGE OF SURGEONS

The United States and Canadian sections of the International College of Surgeons, through the generosity of the Women's Auxiliary, are offering two opportunities to young Canadian or American surgeons.

One is a scholarship of $3,000 to pay for transportation and living expenses for one year for a young surgeon who wishes to study abroad. The doctor selected for the scholarship will be expected to spend at least ten months of the year as a resident or fellow in a teaching center in Europe or South America.

The other is an invitation to two residents or fellows in training in surgery to attend the annual meeting of the college and present a clinical or scientific paper. Transportation and living expenses during the meeting, not to exceed $300, will be provided by the college. The next annual meeting will be in Philadelphia from September 12 through September 15.

Further information concerning both offers can be obtained from the Scholarship Committee, International College of Surgeons, 1516 Lake Shore Drive, Chicago, Illinois.

AMERICAN INSTITUTE OF DENTAL MEDICINE

The next annual meeting of the American Institute of Dental Medicine will take place at the Desert Inn, Palm Springs, California, October 23 to 27. The faculty will consist of Maury Massler, D.D.S., M.S., University of Illinois, Chicago; Valy Menkin, M.D., Temple University Medical School, Philadelphia; Hans Selye, M.D., University of Montreal, Canada; Reidar F. Sognnaes, D.M.D., Ph.D., Harvard School of Dental Medicine, Boston; Wendell L. Wylie, D.D.S., University of California, San Francisco.

All seminar lecturers will participate in a round table forum discussing the application of their subject to the practice of dental medicine. Applications and full information may be secured from the executive secretary, Marion G. Lewis, 2240 Channing Way, Berkeley 4, California.

COURSE IN CLINICAL PATHOLOGY AND PATHOLOGY OF PARASITIC DISEASES

A short, intensive course on the laboratory diagnosis and pathology of parasitic infections will be presented August 15-27 at the Louisiana State University School of Medicine in New Orleans.

The course is designed primarily for pathologists and technologists. However, general practitioners, internists, pediatricians, gastroenterologists and physicians engaged in the practice of public health and tropical medicine who are interested in the laboratory diagnosis of parasitic infections are welcome to attend. The instruction and training will be of assistance to pathologists who are preparing for board examinations, to pathologists and physicians who are responsible for the diagnosis of parasitic infections in their laboratories and to technologists engaged in this specialty.

Registrants should bring their microscopes, equipped with mechanical stages, and their microscope lamps. A limited number of places will be available. The fee for the course is $50.

Persons interested in attending this course may write to: Dr. Clyde Swartzwelder, Department of Microbiology, Louisiana State University School of Medicine, 1542 Tulane Avenue, New Orleans 12, Louisiana.

PAN AMERICAN MEDICAL ASSOCIATION

The next and tenth Inter-American Congress of the Pan American Medical Association will be held in Mexico City. The scientific sessions will begin on March 25, 1957. The Congress will be held in sections covering all branches of medicine and surgery. There will be medical moving pictures, panel discussions and scientific and technical exhibits. The association has forty-two medical sections including the new Section of General Practice.

Four days will be devoted to scientific sessions, and the next three days will be spent in sight-seeing, with visits to Cuernavaca, Taxco and Acapulco. The following week medical meetings will be held in Guatemala City in conjunction with the local chapter there.

The president of the Association is Dr. Pedro A. Gutiérrez Alfaro, Minister of Sanitation and Public Welfare of Venezuela, and the executive director is Dr. Joseph J. Eller, 745 Fifth Avenue, New York, New York.

SURVEY OF POSTGRADUATE MEDICAL EDUCATION

The Council on Medical Education and Hospitals of the American Medical Association has completed a comprehensive survey of postgraduate medical education in the United States. The material collected during the study has been worked into a final report and is being published as a series of special articles in the *Journal of the American Medical Association.* The first study titled "The Scope and Extent of Postgraduate Medical Education in the United States," was published in the February 26 number. Others will appear as follows: March 12, "The Physician as a Lifelong Student. March 26, "The Objectives and Content of Postgraduate Medical Education." April 9, "Educational Methods in Postgraduate Teaching." April 23, "Time and Place Arrangements of Postgraduate Courses for Practicing Physicians." May 7, "Sponsorship and Administration of Postgraduate Medical Education." May 21, "Financing Postgraduate Medical Education." June 4, "The Future of Postgraduate Medical Education."

ART AND HOBBY SHOW AT STATE MEETING

An exhibit of art and hobby projects is to be shown at the Minnesota State Medical Association annual meeting, scheduled for Minneapolis on May 23, 24 and 25. The exhibition will be more inclusive than ever before; not only paintings in various media, photography and sculpture are being shown, but also any hobby projects which are suitable for this type of exhibit. Complete information and entry blanks will be available in April from the State Office, 496 Lowry Medical Arts Building, St. Paul. Any Minnesota doctors doing any of the above type of art or hobby work are encouraged to exhibit. The showing will be appraised by competent judges, and numerous awards will be given. Co-chairmen for this event are Dr. J. S. Milton and Dr. F. A. Zinter, Minneapolis.

PUBLIC HEALTH RADIO-TV SERIES

A thirteen-week series of programs on public health will be presented on stations KSTP and KSTP-TV, Twin Cities, beginning March 9. The programs, which will consist of interviews of public health officials by Bee Baxter, radio and television personality, have been planned in co-operation with the section of public health education of the Minnesota Department of Health.

The interviews will be carried on television (KSTP-TV) on Wednesdays at 1:30 p.m. and on KSTP radio on Thursdays at 11:30 a.m.

Persons to be interviewed on the series include Dr. R. N. Barr, Dr. Helen Knudsen, Dr. Henry Bauer, Dr. Dean S. Fleming, Alberta Wilson, Dorothy Hagland, Dr. A. J. Chesley, Dr. Carl Lundeberg, Dr. A. B. Rosenfield, Frank L. Woodward, Herbert Bosch, Dr. Dale C. Cameron, Marie Ford and Dr. Gaylord Anderson.

MINNESOTA HEART ASSOCIATION

Minnesota institutions and individuals desiring consideration of Grants-in-aid and Fellowships in support of cardiac research and study from the 1955-56 research budget of the Minnesota Heart Association must present their requests on or before May 1, 1955.

Approved grants will extend from July 1, 1955, through June 30, 1956.

Further information may be secured by writing the Minnesota Heart Association, 532 Endicott on Fourth, Saint Paul 1, Minnesota.

THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

F. H. Magney, M.D., Secretary

License of Arthur F. Sether, M.D., Suspended for Three Years

On February 4, 1955, the Minnesota State Board of Medical Examiners suspended for a period of three years the medical license held by Arthur F. Sether, M.D., of Ruthton, Minnesota, because of his violation of the terms of a stipulation which Dr. Sether signed following his personal appearance before the Board on May 18, 1951, in response to a citation for the revocation of his license. In that stipulation, which Dr. Sether entered into with the Board on August 23, 1951, in addition to other provisions he agreed to refrain from the personal use of the so-called barbiturates and alcoholic liquors in every form. It was because of the violation by Dr. Sether of this portion of his agreement with the Board that his medical license was suspended.

Dr. Sether, who was licensed to practice medicine in Minnesota in 1935, was born in St. Paul, Minnesota, on November 5, 1905. After taking a one-year internship at Minneapolis General Hospital, Dr. Sether graduated from the University of Minnesota with an M.D. degree in 1936. In the following year Dr. Sether opened an office for the practice of medicine at Ruthton, where he has practiced since that time.

Woman's Auxiliary

MID-WINTER MEETING WELL ATTENDED

Mrs. L. Raymond Scherer

The Mid-Winter Board meeting of the Woman's Auxiliary to the Minnesota State Medical Association was well attended despite below-zero weather.

The meeting was held February 10 at the Town and Country Club in St. Paul and was attended by county and state auxiliary officers and committee chairmen.

Members were welcomed by the auxiliary president, Mrs. Peter S. Rudie of Duluth. This was followed by the pledge to the flag and the pledge to the auxiliary, led by Mrs. John Dordal of Sacred Heart.

Regular reports of the secretary and the treasurer were read and approved, followed by a report of the president on the meeting of presidents and presidents-elect in Chicago during November of 1954. Mrs. Rudie stated that the meeting offered an excellent chance for an exchange of information with other state auxiliary presidents, and she reported on the many excellent panel discussions, including such subjects as organization, program, publications, *Today's Health,* the American Medical Education Foundation, public relations, legislation, civil defense, mental health, allied medical careers, and finances.

Following Mrs. Rudie's report, members heard a report from the president-elect, Mrs. H. H. Fesler of St. Paul, and the second vice president, Mrs. V. J. Schwartz of Minneapolis. Then followed reports from regional advisors in attendance: Mrs. O. M. Heiberg of Worthington, Mrs. Ralph Creighton of Minneapolis, and Mrs. J. C. Buscher of St. Cloud who reported on activities of auxiliaries in their districts.

State committee chairmen reported on various activities of their committees; reports were heard on Bulletin, legislation, organization, program and health education, public relations, American Medical Education Foundation, health days, newsletter, school of instruction, medical and surgical relief.

County auxiliary presidents in attendance told of individual county auxiliary projects. Reports included were from presidents of the following county auxiliaries: Hennepin, Park Region, Ramsey, Renville-Redwood, St. Louis and Stearns-Benton.

Following the completion of the business meeting, auxiliary members attended the luncheon meeting, presided over by the president. Mrs. Rudie introduced Mrs. V. J. Schwartz, Minneapolis, second vice president, who in turn, introduced Miss Marguerite Breen, Director of Public Relations for the Minnesota Tuberculosis and Health Association. Miss Breen thanked the state auxiliary for its many years of participation in her organization's program of promoting tuberculosis essay contests among high school and junior high school students in Minnesota. She exhibited the annual plaque and trophy given to the best essay on tuberculosis by a junior and senior student each year. The auxiliary sponsors this project jointly with the tuberculosis and health group, and Mrs. Schwartz served as a judge for the 1954 awards. Miss Breen then presented Barbara Sitzman, senior division winner, and Judy Lewis, junior division winner, who presented their prize-winning essays. Both girls are from St. Agnes High School in St. Paul. Auxiliary members agreed that their essays were of excellent quality and fully deserving of the awards they won.

RAMSEY COUNTY AUXILIARY REPORTS ACTIVITIES

Mrs. L. T. Simons

The State Medical Auxiliary Mid-Winter Board meeting was held on February 10, 1955, at the Town and Country Club, St. Paul. Mrs. W. H. Von Der Weyer and Mrs. B. E. O'Reilly were in charge of hospitality. Ramsey county members present were: Mrs. W. P. Gardner, president, Mrs. H. O. Peterson, president-elect, Mrs. Charles Waas, Mrs. C. W. Froats, Mrs. J. L. Benepe, Mrs. H. H. Fesler, Mrs. Mark Ryan and Mrs. John Ryan. Mrs. P. K. Arzt, chairman of state and Ramsey county legislation, presented both her own report and Mrs. E. M. Hammes' concluding report on the Dr. William A. O'Brien fund.

The Auxiliary took part in the Inter Club Council's "What's Cooking?" program held in the Lowry Hotel on March 28, as its public relations project for the year. Mrs. J. F. Madden and Mrs. J. R. Meade, auxiliary representatives, organized the medical scholarship material used on the education panel. Dr. Charles Rea, St. Paul, a member of the panel, spoke on medical scholarships and their allied fields. Mrs. O. I. Sohlberg served in an advisory capacity on the planning of the legislation panel.

Mrs. R. O. Burmeister, philanthropic chairman, reported a total of 175 cancer dressings. The Auxiliary assists this committee by making dressings during and following the board meetings.

The Medical and Surgical Relief Committee, Mrs. H. F. Schroeckenstein, chairman, packed 29 cartons of supplies in January. These were sent to the Episcopal Mission Orphanage in Osaka, Japan.

Mrs. R. F. Sturley, nurse recruitment chairman, reported her committee has been busy arranging for transportation of nurses to Murray, Roosevelt and Mahtomedi High Schools during February. These nurses talk to junior and senior girls who are interested in nursing as a career.

In Memoriam

ALOYSIUS STEPHEN FLEMING

Dr. A. S. Fleming, formerly of Minneapolis, died January 19, 1955, at Madison, N. J., where he had been living since his retirement in 1950. He was eighty-three years old.

Dr. Fleming was born in Eau Claire, Wisconsin, in 1872. He attended the University of Wisconsin for two years and got his medical training at the medical college of Hamline University. For fourteen years, he practiced in Wheaton, Minnesota, before coming to Minneapolis in 1913.

He was a former member of the staff of Hillcrest Hospital and was a member of the Hennepin County Medical Society, the Minnesota State Medical Association, and the American Medical Association. He was also a "Fifty Club" member of the Minnesota State Medical Association.

Surving are three sons, Noel C., Madison, N. J.; Dr. Dean S., Hopkins, and Allan W., Duluth; a daughter, Helen Fleming, Madison, N. J., and four grandchildren.

CHARLES HARRY GHENT

Dr. C. Harry Ghent, St. Paul physician, died January 31, 1955, in St. John's Hospital, St. Paul. Dr. Ghent had been ill for four months. He was sixty-six.

Dr. Ghent was born in Centralia, Illinois, in 1888. He graduated from the Northwestern University Medical School in 1911 and interned at Passavant Hospital in Chicago. He practiced in Dawson, Minnesota, and White Rock, South Dakota, before moving to St. Paul in 1916.

He had been a member of the staff of St. John's Hospital since 1916 and was also on the staff of Mounds Park Hospital. He was a member of the Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Dr. Ghent is survived by his wife, Florence; a daughter, Mrs. John B. Rutledge of Chicopee, Massachusetts; a sister, Mrs. John B. Hamilton, Carbondale, Illinois; and three grandchildren.

CHARLES ALBERT HALLBERG

Dr. Charles A. Hallberg, Minneapolis physician and surgeon, died suddenly January 30, 1955, at St. Barnabas Hospital. He was sixty-seven.

Dr. Hallberg was born in Stockholm, South Dakota. He received his medical degree at the University of Illinois in 1911 and interned at University Hospital in Chicago.

In 1917, Dr. Hallberg went to the Mayo Clinic, Rochester, Minnesota, on a fellowship. Except for a period of service during World War I, he remained there until entering private practice. He became an associate in medicine on the Mayo Clinic staff and took his specialist training in surgery there. He entered private practice in Minneapolis in 1926.

A staff member of St. Barnabas and the Northwestern Hospitals, Dr. Hallberg was a fellow of the American College of Surgeons, a member of the American Medical Association, the Minnesota State Medical Association and the Hennepin County Medical Society and the Minneapolis Surgical Society.

He is survived by two sisters, Mrs. Alma Darud, Seattle, Washington, and Mrs. Amanda Oestrich, La Bolt, South Dakota.

RAY GEORGE JOHNSON

Dr. Ray G. Johnson, Stillwater physician, died January 14, 1955. He was fifty-nine years old.

Dr. Johnson was born in Waltham, Minnesota, in 1895. He obtained a B.S. degree from Carleton College in 1920 and his M.D. degree from the University of Minnesota Medical School in 1924.

Following his internship at Mounds Park Hospital in St. Paul, Dr. Johnson practiced in St. Paul until 1938. In 1939, he became resident physician at the Minnesota State Prison at Stillwater.

He was a member of the Washington County Medical Society, the Minnesota State Medical Association, the American Medical Association, St. John's lodge No. 1, and A. F. and A. M.

He is survived by his wife, Ida May; two daughters, Barbara and Janet Rae; one brother, Vaughn, of Terry, Montana; two sisters, Mrs. Howard Crowther, Saint Paul and Mrs. Mabel Jordre of Oberon, North Dakota.

GEORGE P. KIRK

Dr. George P. Kirk, physician of East Grand Forks, Minnesota, died on January 20, 1955, at the age of eighty-one.

Dr. Kirk was born in Minnesota in 1874. He graduated from the University of Minnesota Medical School in 1896.

In 1949 Dr. Kirk, a member of the Red River Valley District Medical Society, became a life member of the Minnesota State Medical Association. He had been a member of the State Association for forty years.

During his active life, Dr. Kirk had been a health officer and city doctor. He was on the staff of St. Michael's and Deaconess Hospitals.

Dr. Kirk is survived by his wife, Anna; two sons, Cyrus of Minneapolis and Paul of Edmonton, Alberta, Canada; one daughter, Mrs. Edward Kuppich of Fargo; a brother, John H. of Glendale, California; a sister, Mrs. Ernest Freivogle of Richfield, Connecticut; and seven grandchildren.

Of General Interest

On February 3, two hundred Winnebago citizens turned out to honor the forty-seven years of service of **Dr. M. D. Cooper.** Dr. and Mrs. Cooper were guests of honor at a testimonial dinner sponsored by the Winnebago Community Club in the Presbyterian Church.

"This Is Your Life" theme was carried out, with many citizens and friends giving short tributes to the doctor's contribution to the life and welfare of the community. **Dr. Ralph Armstrong,** president of the Blue Earth Valley Medical Society, commended Dr. Cooper's long years of service.

In accepting the many honors, Dr. Cooper pointed out that when he arrived in Winnebago it was a thriving town, that there were several industries, three hotels and a college. "However," he said, "it was not these things that brought me here. It was the people."

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Dr. Philip S. Hench, Rochester, presented a talk entitled, "X Marks the Spot," at a program in St. Paul on February 17, sponsored by the Rochester Area, Women's Association, Minnesota Historical Society.

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Principal speaker at a meeting for heart fund volunteers in Anoka on February 16 was **Dr. Morley Cohen,** Minneapolis, who spoke on "Recent Advances in Treatment of Heart Disease in Children."

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Dr. R. H. Puumala, of Cloquet, attended a postgraduate course in internal medicine at the University of Minnesota Continuation Center in mid-February.

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At the annual meeting of the Fosston Hospital Association in Fosston on February 8, **Dr. George Sather** gave a talk on different types of hospital and medical insurance policies.

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It was reported early in February that the Westbrook Commercial Club was hoping to locate and persuade another physician to open a practice in or near Westbrook. The community now has one physician.

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Dr. George S. Allen, Mineral Springs Sanatorium, Cannon Falls, attended the Veterans Administration Army-Navy Conference in Atlanta, Georgia, early in February. Subject of the conference was the chemotherapy of tuberculosis.

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Dr. and Mrs. Edward D. Detjen, Bigfork, spent the first week of February in Texas, visiting relatives and absorbing sun.

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Principal speaker at the annual meeting of the Citizens Mental Health Association at Minneapolis on February 18 was **Dr. Adelaide M. Johnson,** Rochester, who discussed problems of bringing up children in modern society.

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Dr. Walter A. Fansler, Minneapolis, took part in a symposium presented at Detroit on February 9 before members of the Michigan Academy of General Practice and the Wayne County Medical Society. Subject of the symposium was problems of the general practitioner.

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Dr. Tauno Ketola, staff member of the Cokato Hospital since September, 1954, was called into service with the Air Force at Montgomery, Alabama, at the end of January.

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Dr. William S. Chalgren, Mankato, spoke at a meeting of the Windom Kiwanis Club on February 8 on the subject, "A Psychiatrist's Approach Towards Juvenile Delinquency."

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Dr. and Mrs. A. B. Rosenfield, Minneapolis, and **Dr. and Mrs. C. L. Sheedy,** Austin, left on February 11 for a six weeks' vacation on various Caribbean Islands. At San Juan, Puerto Rico, both physicians presented papers before medical groups.

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On February 15, **Dr. A. J. Lenarz** left his practice at Browerville for a residency in surgery at St. Joseph's Hospital, St. Paul. He expects to return to Browerville on July 1.

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Dr. Harris P. Hinderaker, Bird Island, was the principal speaker at a meeting of the Bird Island Parent-Teachers Association on February 7. He spoke in connection with the local heart fund drive.

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A radio talk was given by **Dr. F. H. Koenecke,** Lakefield, over station KWOA, Worthington, on February 6. His talk was on "Premature Babies" and was part of the program entitled, "Your Health Hour," sponsored by the Southwestern Minnesota Medical Society and Auxiliary.

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After two years in the Air Force **Dr. R. J. Lindeman** returned to his practice at Paynesville in mid-January. While in service he was stationed at Colorado Springs, Colorado.

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Dr. M. J. Schirber, Grand Rapids, was the principal speaker at a meeting of the Itasca County Historical Society in Grand Rapids on February 7. He discussed medical instruments and methods used by pioneer physicians in Itasca County.

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Dr. L. G. Flanagan, Austin, attended a continuation course in ophthalmology at the University of Minnesota during the first week of February.

Dr. Nicholas H. Zeller, staff member of the Worthington Municipal Hospital since April, 1954, left in mid-January for a two-year tour of duty in the Army.

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Dr. Dean S. Fleming, director of disease prevention and control for the Minnesota Department of Health, has announced that more than 100,000 children in Minnesota may be vaccinated for poliomyelitis this spring. The decision about the vaccination program depends upon the results of a report on the Salk vaccine to be issued at the University of Michigan about April 1. If the report is favorable, Minnesota would accept a supply of the vaccine from the National Foundation for Infantile Paralysis and make it available to physicians throughout the state.

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Dr. Frederic Kottke, head of the department of physical medicine and rehabilitation at the University of Minnesota, was the principal speaker at a meeting of the Minneapolis District Multiple Sclerosis Society on January 18. Dr. Kottke discussed the university's rehabilitation center.

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Dr. C. J. Swendseen, Graceville, arrived in Starbuck in mid-January to take charge of the practice of **Dr. A. F. Giesen,** who was hospitalized at Northwestern Hospital, Minneapolis, due to injuries suffered in a fall.

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The Swedish consul general in Minneapolis announced in mid-January that **Dr. Henry P. Linner** had been decorated by King Gustav of Sweden. Dr. Linner was made a knight of the Order of North Star. He was one of four Twin Cities men honored for achievements in their fields and for their contributions to Swedish-American relations.

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Dr. I. L. Oliver, Graceville, was the guest speaker at a meeting of the Graceville Nurses Association on February 10. He spoke on new methods in heart and lung surgery and new types of anesthesia and sutures.

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The former Mayo Institute of Experimental Medicine, located near Rochester, has been renamed the **Institute Hills Farm.** The change was made because most of the research laboratories of the institute have been moved to the Medical Sciences Building in Rochester. The farm facilities now are used primarily for breeding and holding animals for research.

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The main speaker at a meeting of the Exchange Club in Cloquet on January 24 was **Dr. Kenneth W. Douglas,** superintendent of Sandstone State Hospital. He discussed the problems of alcoholism and narcotic addiction.

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Dr. and Mrs. H. L. Smith left Rochester on January 31 for a trip to Europe. They plan to return in May. Dr. Smith is a retired member of the Mayo Clinic staff.

Dr. Shohei Shirai of the Mesaba Clinic, Coleraine, spoke on the subject of personal health at a meeting of the Women's Club at Bovey late in January.

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Among the speakers at a conference of the Twin City Preschool Education Association at the University of Minnesota on January 22 was **Dr. Edwin Burklund,** St. Paul pediatrician, who spoke on the value of nursery schools.

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Dr. Lewis Hanson, Frost, was elected chairman of the Faribault County Public Health Nursing Advisory Board at the annual meeting of the board in Blue Earth in mid-January.

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Dr. John E. Hildebrand, Bemidji, suffered a severe fracture of one leg when he fell while crossing an icy street in Bemidji in mid-January. His practice was conducted during his convalescence by his associate, **Dr. J. K. Hartjen.**

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Main speaker at the January meeting of the Mankato Association for Retarded Children was **Dr. John Olive,** pediatrician at the Mankato Clinic. His talk was primarily on the Rh factor and kernicterus.

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Dr. Mario Fischer and **Dr. Edward Zupanc,** both of Duluth, were among the new directors appointed to the board of the Duluth Mental Hygiene Clinic at a meeting late in January.

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Common illnesses and practical ways of dealing with them were discussed by **Dr. Viktor O. Wilson,** Rochester-Olmsted County health director, at a public meeting in Rochester late in January. The talk was one of a series on family health being given in the community. Other lecturers in the series included **Dr. R. M. Wilder** on nutrition and **Dr. George Williams** and **Dr. R. L. Faucett** on the development of mental and emotional health.

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Dr. James J. Coll, Duluth, was guest speaker at a meeting of the Arrowhead Society of Medical Technologists at Duluth on January 28.

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Three Twin Cities physicians took part in a discussion of "Surgery of the Heart" on a television program on WCCO-TV on January 30. They were **Dr. Richard Varco, Dr. J. Arthur Myers, Dr. John Briggs** with Mr. Thomas Mulrooney.

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Dr. H. C. Otto, Frazee, who was operated upon at the University of Minnesota Hospitals in November, returned to his home in Frazee late in January. He was not expected to resume his practice for some time.

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"Health and the Child" was the title of an address by **Dr. John K. Butler,** Cloquet, at a meeting of the Parent-Teacher Association in Carlton on January 18.

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The Truman Public School Board has appointed

Dr. M. J. Lester, Truman, to fill the unexpired term of a late member of the board. The term runs until July 1.

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Dr. Russell M. Wilder, emeritus member of the Mayo Clinic staff, Rochester, was honored at a meeting of dietitians and nurses in Rochester late in January. Dr. Wilder recently won the American Medical Association's Goldberger award in clinical nutrition. He is best known for his studies of diabetes mellitus, carbohydrate metabolism and a program for vitamin-enriched bread and flour.

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Dr. S. T. Normann, Waseca, has been named coroner of Waseca County, replacing **Dr. Orvie J. Swenson,** who held the office for twenty-six years.

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Among the speakers at a meeting of the Minnesota Tuberculosis Nursing Council in St. Paul on January 14 was **Dr. F. F. Callahan,** St. Paul, chief of the division of medical services of the Minnesota Department of Public Welfare. He discussed proposed tuberculosis legislation in the state.

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At a meeting of the Dennison village council in January, **Dr. A. M. Nielsen,** Northfield, was named official physician for the village.

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Dr. Ivan D. Baronofsky, associate professor of surgery at the University of Minnesota Hospitals, has joined the Navy for a two-year tour of duty. He reported for service in February with the rank of lieutenant-commander.

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Dr. Royden H. Belcher, Wheaton, was the guest speaker at a meeting of the Graceville Nursing Association at Graceville in January. He spoke on new treatment methods in various types of epilepsy.

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Little-known medical services in the community were described and explained by **Dr. G. M. B. Hawley** at a meeting of the Red Wing Kiwanis Club in January. Among the subjects he discussed were the grievance committee of the Goodhue County Medical Society, surgical conferences, pathology examinations, and hospital services.

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The Duluth Chapter, Minnesota Academy of General Practice, held a special refresher course on "Fluid and Electrolyte Balance" at St. Luke's Hospital, Duluth, on February 18. Four Minneapolis physicians presented talks during the afternoon and evening sessions. **Dr. John LaBree** and **Dr. Richard J. Frey** discussed "Electrolyte Balances in Cardiac and Renal Disease," and **Dr. W. P. Eder** and **Dr. Richard Raile** talked on electrolyte balance in regard to the surgical and pediatric fields.

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Dr. H. F. Helmholz, Rochester, emeritus member of the Mayo Clinic staff and president of the Olmsted County Community Council, was the principal speaker at the annual dinner meeting of the Rochester Community Chest on January 19.

One week after **Dr. John W. Ekblad,** Duluth, retired as St. Louis County coroner on January 1, he fell down an outside stairs while calling on a patient and sustained a hip fracture. During his sixteen years as coroner, which often required strenuous activity, he was never injured. Dr. Ekblad is eighty years of age.

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Dr. Sidney Esensten, Minneapolis, has received a commendation medal for meritorious service while a prisoner of war in Korea from November, 1950, to September, 1953. The commendation praised Dr. Esensten's efforts to render effective aid to sick and wounded comrades in spite of the difficulties imposed by his North Korean captors.

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Dr. George R. Smith has returned to Hutchinson after two years of active duty in the Army. He has resumed his medical practice with **Dr. Elmer W. Lippmann** and **Dr. Dan W. Huebert.**

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Dr. T. S. Eberley, Benson, sustained a severe leg fracture when his car skidded on an icy highway near Benson about the first of the year and overturned, hurling him from the car. He was taken to Miller Hospital in St. Paul for treatment.

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Dr. William B. Halme, who closed his practice in Wadena nearly two years ago to enter the armed forces, has leased a recently vacated building in Wadena. He expects to be discharged from service before June 1 and plans to resume his practice at Wadena immediately.

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Early in January, **Dr. F. W. Calhoun,** Albert Lea, was honored on his seventy-eighth birthday at a surprise party given by friends. Dr. Calhoun is now in his forty-eighth year of practice, all in the Albert Lea area.

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It was announced in January that **Dr. Herman Miller,** Grand Meadow's only physician, had been called into service with the Air Force.

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Dr. John F. Briggs, St. Paul, has been elected to the Horseshoe Club of London, England, an organization whose object is to foster friendship between American, Canadian and English men and women interested in the cure and prevention of disease.

Dr. Briggs has also been asked to act as a collaborator for *Medico-Surgical Annals,* a new magazine which will stress diseases of the chest and thoracic surgery. The magazine will be published in Spain.

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Dr. Gordon R. Kamman, St. Paul, spoke on "Psychological Evaluations in Medical Practice" at a meeting of the Sixth District Medical Society at Bismarck, North Dakota, on February 22.

NEW LOCATIONS

Dr. Carl Anderson, formerly of Miles City, Montana, has been appointed resident physician at the Minnesota State Penitentiary at Stillwater.

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Dr. Carl Heinzerling, formerly of Watertown, has opened offices for the practice of medicine in Chaska.

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Dr. A. L. Ourada, Ceylon, has opened an office at Fairmont. He now maintains office hours in Fairmont in the morning and in Ceylon in the afternoon. He expects to move to Fairmont in June.

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Dr. Edward Menefee, who recently completed a residency in pathology at Rochester, is now associated with the Naeve Hospital, Albert Lea, as a pathologist.

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Dr. John Meyers, formerly of Chicago, has become associated in practice with **Dr. L. J. Monson** in Canby.

MINNESOTA BLUE SHIELD-BLUE CROSS

Blue Cross News

During the month of December, 1954, 18,870 participant subscribers were enrolled in Blue Cross bringing the net enrollment as of December 31, 1954 to 1,000,051 participant subscribers.

The amount of hospital care benefits received by Minnesota Blue Cross subscribers during 1954 amounted to $18,272,129.50, an increase of $2,822,556.55 over the amount provided in 1953.

During the past year, 165,989 Blue Cross subscribers were hospitalized for a total of 1,003,552.3 days of care. Average length of stay in the hospital for a Blue Cross patient was six days.

The four major causes for hospitalization of Minnesota Blue Cross subscribers during 1954 were: pregnancies, injuries and accidental poisonings, respiratory ills and digestive illness. There were 28,579 pregnancy cases of which 5,612 were for pyelitis of pregnancy, toxemias, ectopic, et cetera, and 22,967 were deliveries amounting to over 2½ million dolllars in benefits. There were 25,306 injuries and accidental poisonings amounting to over one million dollars in savings to subscribers. Over 1½ million dollars in benefits was provided for 22,703 subscribers who had respiratory illnesses. 21,996 subscribers had digestive ailments of which 11,600 were surgical cases. A total of approximately $3,200,000 was paid in benefits for digestive diseases alone.

Since 1933 when Blue Cross was first started here in Minnesota, 1,729,994 subscribers have spent 10,479,006.6 days in hospitals and have received benefits amounting to $110,949,752.

Blue Shield News

In the year 1954, payments to doctors for services rendered to Blue Shield subscribers totaled $4,970,000. This amount marks a new high in the annual amount of money paid to doctors. For the year 1954, payments have been over $700,000 more than was paid in 1953. Further analysis shows that the payments made in 1954 are 16 per cent greater than the amounts paid in the preceding year, 1953. The dollar increase in 1954 was due principally to the increased benefits which became effective on January 1, 1954, but also to the increase in the number of services rendered by doctors of medicine.

Another interesting factor in the growth of Blue Shield is revealed in the compilation of data which discloses that during 1954, 169,445 services were rendered by doctors for which Blue Shield provided allowances. This can be compared with a total 148,561 compensable services rendered during the year 1953.

Internal changes have also been made in the Blue Shield organization in order to provide more time to disseminate information to members of the medical profession regarding the Blue Shield contract and procedures. These changes have been of some help in the doctors' offices and have led to greater efficiency in Blue Shield. In this particular field, many of the problems which have formerly existed were eliminated through professional relations activities in 1954. This program is continuing and is being expanded as rapidly as possible.

Blue Shield enrollment as of December 31, 1954, totaled 670,757 participant subscribers. Enrollment as of December 31, 1953, was 609,474 participant subscribers. This represents slightly more than an 11 per cent increase in enrollment throughout the year 1954.

ATHLETIC ACCIDENT BENEFIT PLAN

(Continued from Page 183)

Control approved an increase to $3 per year for the boy who participates in football alone or football and other sports, and $1.50 per year for the athlete not playing football. It is hoped and intended that this will take care of the increased payments.

Your medical service committee and H. R. Peterson realize that there are a number of problems still to be solved and that there always will be administrative and public relations items to be considered. I assure you that utmost co-operation can be expected from the league and its medical advisor, and improvements will continue to be made. Comments and discussions from the physicians of Minnesota are more than welcome at any time and at all times.

Appreciation is expressed at this time to Dr. J. A. Bargen, chairman of the Medical Service Committee of the Minnesota State Medical Association, and his committee for their energetic help and advice in the 1954-1955 revision of this program.